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EDITORIAL

THE STAFF OF THE SECTION BY THE ARMED FORCES AND PUBLIC HEALTH SERVICE

THE new department of the JOURNAL, the section by the Armed Forces and Public Health Service, was announced in the February issue. Now we want to acquaint our readers with those in charge.

ARMY

Major General James M. Epperly is the advisor from the Army. He was born in Huntsville, Missouri, and studied at the University of Missouri and St. Louis University, where he received his D.D.S. degree. His military career started in World War I. Commissioned a first lieutenant, he was assigned to Fitzsimons Army Hospital, where he served as dental intern. His first overseas assignment was to the Philippines. Just before World War II he was sent to West Point and then, as a lieutenant colonel, to Camp Breckinridge. Here he assumed the duties of chief of dental service and was promoted to the rank of colonel. He moved with the Ninth Army to Europe. In 1953 he was on temporary duty in Greece and was made a brigadier general. On Dec. 1, 1956, he was promoted to major general and was appointed assistant surgeon general and chief of the Army Dental Corps. He has received the following decorations: Bronze Star Medal, Legion of Merit, Army Occupation Medal (Germany), American Defense Service Medal, American Campaign Medal, European-African Middle Eastern Campaign Medal, Army Commendation Ribbon with cluster, World War II Victory Medal, World War I Victory Medal, and National Defense Service Medal.

MAJOR GENERAL JAMES M. EPPERLY

The associate editor from the Army is *Colonel Joseph L. Bernier.* He was born in Chicago, Illinois, and received his D.D.S. and M.S. degrees from the University of Illinois. In 1949 he received the F.D.S. from the Royal College of Surgeons of England. Colonel Bernier is professor of pathology at Georgetown University; director of the Dental and Oral Pathology Division, Armed Forces Institute of Pathology in Washington, D. C., and pathologist to the Registry of Oral Pathology of the American Dental Association. He has received the following decorations: commandership in the Order of the Crown of Italy, Meritorious Service Medal, Army Occupation Medal, Army Defense Medal, American Campaign Medal, World War II Victory Medal, Korean Victory Medal, European African Medal, Middle East Service Medal, and National Defense Medal. He received the Louis Livingston Award, the Tufts University School of Dental Medicine Award, the Thomas Hinman Award, the John R. Callahan Award, and the Alfred C. Fones Award. Colonel Bernier is a diplomate of the American Board of Oral Pathology, the American Board of Periodontology, and the American Board of Oral Medicine.

COLONEL JOSEPH L. BERNIER

NAVY

Rear Admiral Curtiss W. Schantz is the advisor from the United States Navy. He was born in Cottage Grove, Wisconsin. He studied at the University of Wisconsin and Marquette University, from which he received the D.D.S. degree. After practicing dentistry, he was appointed to the dental corps of the United States Navy and was commissioned lieutenant junior grade in 1926. He served in various places, including the Brooklyn Navy Yard, the U.S.S. Chicago, and the Naval War College. From 1936 to 1937 he took a graduate course in oral surgery at Columbia University; then he joined the U.S.S. Augusta, flagship of the United States Asiatic Fleet. During this assignment he did graduate work as a German language student in Shanghai and later visited the U.S.S.R. with the Yarnell Mission. During the Sino-Japanese hostilities, he was aboard the U.S.S. Augusta and served as senior dental officer at the United

REAR ADMIRAL CURTISS W. SCHANTZ

States Navy Yard, Cavite, Philippine Islands. During the battle of Pearl Harbor he was aboard the U.S.S. Nevada. He was attached in 1943 to the Naval Training Center in San Diego, California; in 1945 he was attached to the staff of Commander, Service Force, Seventh Fleet, and later to the staff of Commander, Philippine Sea Frontier. He then was ordered to the Naval Hospital at Great Lakes, Illinois, as chief of the dental service. During the Korean War he served as a member of the Pacific Fleet Evaluation Board under Admiral A. W. Radford. In 1953 he returned to the United States and served as command dental officer and dental officer for the Naval Academy, after which, on Nov. 15, 1954, he became commanding officer of the Dental School, National Naval Medical Center, Bethesda, Maryland. He served here until he was advanced to the rank of real admiral with the duties of assistant chief for dentistry, Bureau of Medicine and Surgery, Washington, D. C.

Rear Admiral Schantz is a Fellow of the American College of Dentists, a diplomate of the American Board of Oral Medicine, and a Fellow of the American Association for the Advancement of Science. He is a Companion of the Naval Order of the United States.

He has been awarded the Commendation Ribbon for the Navy Unit. He has the Navy Expeditionary Medal (Panay Incident), the China Service Medal, the American Defense Service Medal with star, the American Campaign Medal, the Asiatic-Pacific Campaign Medal with two stars, the World War II Victory Medal, the National Defense Service Medal, the Korean Service Medal, the United Nations Service Medal, and the Expert Rifleman Medal.

Captain Theodore A. Lesney is our associate editor from the Navy. He is a 1937 graduate of the Northwestern University School of Dentistry in Chicago, Illinois. Captain Lesney is a diplomate of the American Board of Oral Surgery and a Fellow of the American College of Dentists. He served as a consultant instructor to intern and residency training programs and had teaching experience as a member of the faculty of Northwestern University as well as at the United States Naval Dental School, National Naval Medical Center, Bethesda, Maryland. He is now chief of dental service, United States Naval Hospital, San Diego, California.

CAPTAIN THEODORE A. LESNEY

AIR FORCE

Major General James S. Cathroe is our advisor from the Air Force. He was born in Omaha, Nebraska, and after graduating from Technical High School, entered Creighton University Dental School as a member of the

MAJOR GENERAL JAMES S. CATHROE

R.O.T.C. After graduation he was selected for an Army dental internship at Letterman General Hospital. He entered the regular service and shortly afterward was appointed to Fort Niagara as dental surgeon of the Fort. In 1931 he was ordered to Washington where he took a course in advanced dentistry at Walter Reed Army Hospital. He served at Schoefield Barracks in Hawaii and at Fort Lewis, Washington. In 1938 he joined the dental staff at the United States Military Academy and then was transferred to Camp Stewart in Georgia as camp dental surgeon. In 1942, when he attained the rank of lieutenant colonel, he was sent to Maxwell Field in Alabama. Here he was assigned to Headquarters Eastern Flying Training Command as the command dental surgeon. In 1949 Colonel Cathroe became command dental surgeon of the Caribbean Air Command, and after that he was base dental surgeon at Lackland Air Force Base. During his tour in Panama he was transferred from the Army to the newly established medical service of the Air Force. He was made command dental surgeon of the Air Training Command, and in 1955 he was promoted to brigadier general and deputy assistant for dental services, Office of the Surgeon General, United States Air Force. Quite recently he was promoted to the rank of major general.

General Cathroe enjoys fishing and hunting and likes Chinese and Italian cooking. His decorations include the American Defense Medal, the World War II Victory Medal, the American Theatre Ribbon, and the National Defense Service Medal.

Colonel Donald C. Hudson is the associate editor from the Air Force. He graduated from the University of Iowa and interned at the Fresno County Hospital. After general practice and an instructorship in the Dental College of the University of Iowa, he entered the regular Army during World War II and did service in France and Germany in 1945 and 1946. He returned to Europe in 1948 as command dental surgeon, United States Air Force, Europe. Later, in 1950, he entered research activity at the National Bureau of Standards.

COLONEL DONALD C. HUDSON

Colonel Hudson was appointed chief of the Dental Sciences Division, School of Aviation Medicine, United States Air Force.

in 1956, a position he holds at present. He is a Fellow of the American College of Dentists and a member of the American Association for the Advancement of Science. His military decorations include the medal of the Legion of Merit.

PUBLIC HEALTH SERVICE

Dr. John W. Knutson, assistant surgeon general and chief dental officer of the Public Health Service, is another of our advisors. He was born in Minneota, Minnesota, and graduated from the Minnesota School of Dentistry with the degree of D.D.S. Following this came a dental internship at the United States Marine Hospital in Chicago. He also graduated from Johns Hopkins School of Hygiene and Public Health with the degree of Master of Public Health, and one year later he received the Doctor of Public Health degree. He served as instructor of biostatistics at Johns Hopkins University, which he combined with graduate studies in public health. Later he was appointed to the National Institute of Health in Minneapolis, where he carried on dental research until he became chief of the Dental Health Section, States Relations Division in 1944 and chief of the Division of Dental Public Health in 1948. Dr. Knutson became chief dental officer of the Public Health Service in 1952.

DR. JOHN W. KNUTSON

Dr. Knutson is a diplomate of the American Board of Dental Public Health. He is also vice-president of the American Dental Association. He has received the University of Minnesota Outstanding Achievement Medal and has been elected honorary member of the All India Dental Association and the Norwegian and Danish Dental Association. Besides this, he is an Honorary Fellow of the Royal Society of Health, England, and the International College of Dentists.

Dr. Edward J. Driscoll, our associate editor from the Public Health Service, is stationed at the National Institute of Health in Bethesda, Maryland. He was born in New Orleans, Louisiana, and received his predental education at Loyola University and the Loyola College of Dentistry, where he graduated with the degree of D.D.S. He interned at the United States Public Health

Service Hospital in New Orleans. After graduate training in anesthesiology at the Doctors Hospital in Washington, D. C., he took the oral surgery course at the Graduate School of Medicine of the University of Pennsylvania. He served as chief of oral surgery at the Manhattan Training Station (United States Coast Guard) from 1942 to 1946. Then he became chief of the Oral Surgery Interne Training Program of the United States Public Health Service Hospital in New Orleans. At present he is chief of oral surgery at the National Institutes of Health in Bethesda, Maryland. Dr. Driscoll is a member of the American Society of Oral Surgeons.

K. H. T.

DR. EDWARD J. DRISCOLL

OPERATIVE ORAL SURGERY

HEMORRHAGE IN ORAL SURGERY

Richard J. Antioch, A.B., D.D.S., Philadelphia, Pa.

SINCE the beginning of time, man has been confronted with the problem of bleeding. In the practice of oral surgery control of hemorrhage is a serious consideration, for bleeding may lead to one of the most distressing complications.[1] For the average patient, it is an alarming experience to be weakened by loss of blood and upset with worry, although it is not serious in every instance. Since the oral surgeon is dealing with bony and soft tissues in an area where free access may not be possible, hemorrhage may obscure the field and make the operative procedure more difficult. In oral surgery the extent of hemorrhage has been vastly underestimated. The blood loss in the average patient undergoing extraction of teeth and alveolotomy compares with blood losses encountered in major surgery.[2] The oral surgeon must anticipate postoperative bleeding before instituting treatment, since any extraction or surgical procedure may give rise to a potential hemorrhage.[3]

Man has been in constant search of the ideal agent or method for the control of hemorrhage. Attempts to control the flow of blood are as old and as interesting as the history of surgery itself. This is revealed in Birch's[4] brief history of hemorrhage. Birch states that during a period of seventy years following 1848 more than 100 agents and various methods were used in the attempt to arrest bleeding.

THEORY OF COAGULATION

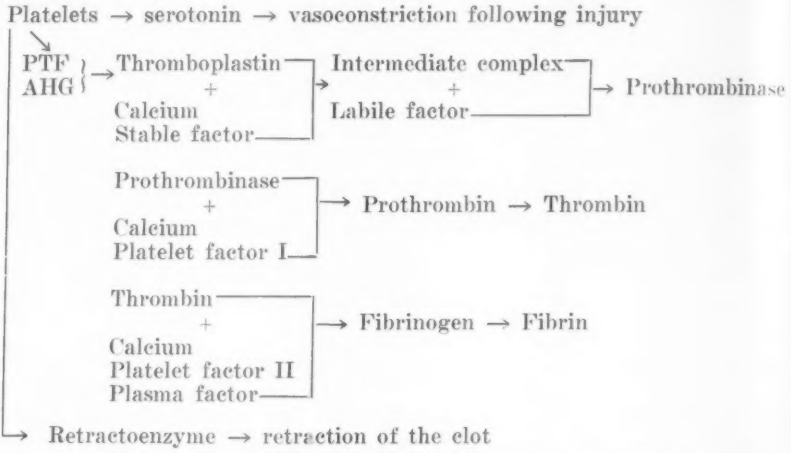
The clotting of blood is a process which protects the patient from excessive loss of blood. Most of Howell's accepted theory of blood coagulation still remains with us today. This classical formula is as follows:

$$\text{Prothrombin} + \text{calcium} + \text{thromboplastin} \rightarrow \text{thrombin}$$
$$\text{Fibrinogen} + \text{thrombin} \rightarrow \text{fibrin}$$

Investigators have been searching for a detailed explanation of this formula of blood coagulation. Various modifications of the original formula have been made.

From the Oral Surgery Section, University of Pennsylvania.

Stefanini[5] gives us a working knowledge of the hemostatic mechanism, according to the latest ideas, in the following series of events:



The diagram begins with the action of an injured vessel producing a temporary, localized vasoconstriction by the autonomic nervous system. The velocity of the blood is reduced, aiding the formation of the clot. Fibrin needles are formed; then the platelets begin to agglutinate at the site of the injury. The agglutination and fibrin formation produce an enmeshment of the red and white corpuscles and platelets. This is followed by the disintegration of the platelets releasing serotonin which produces a prolonged vasoconstriction; then the process of blood coagulation begins. With the lysis of the platelets, thromboplastinogenase or platelet thromboplastic factor (PTF) reacts with thromboplastinogen or antihemophiliac globulin (AHG) to form thromboplastin. The thromboplastin reacts with calcium and the stable factor, convertin, to form an intermediary complex which, in turn, reacts with the labile factor, accelerin, to form a prothrombin-converting factor or prothrombinase. In the presence of calcium, prothrombinase converts prothrombin into thrombin with the aid of a platelet factor. The thrombin, in the presence of an optimal amount of calcium, plasma factor, and a platelet factor with fibrinogen, forms the fibrin clot. The retraction of the clot takes place due to the physical action or retractoenzyme of the platelets.

The platelets are furnished by the reticular cells in the bone marrow. The disintegration of platelets is a prerequisite for production of thromboplastin and, therefore, for the clotting of blood. The lysis of platelets produces thromboplastinogenase which acts upon thromboplastinogen, the inactive form of thromboplastin. The thromboplastin is thought to be present in the plasma as thromboplastinogen.

Thrombin is not ordinarily found in circulation. It must be created when needed; otherwise, the blood would clot at any time. It does not appear in

circulating blood as thrombin but as prothrombin. Thrombin is believed to be the coagulating enzyme, and its concentration in the plasma is a controlling factor in coagulation.

Prothrombin is found in the blood plasma and is produced by the liver. Vitamin K is necessary for its production, and there must be some vital liver function.

Thromboplastinogenase is produced by the lysis of platelets. It is the platelet factor in coagulation which is responsible for the acceleration of thromboplastinogen to thromboplastin.

Convertin exists in the plasma as proconvertin and is a stable factor.[6] Thromboplastin activates proconvertin with calcium, forming an intermediate complex. The intermediate substance reacts with accelerin,[7] a labile factor, producing prothrombinase. Accelerin is thought to be present in circulation as inactive pro-accelerin until activated for coagulation.

The fibrin, which is the insoluble protein, forms the basis of the clot; it does not exist in the circulating blood as fibrin but as soluble fibrinogen. Fibrinogen is a plasma protein found in the blood and formed in the liver. Production of fibrinogen will be decreased to such an extent in liver disease that hemorrhage will be excessive.

This is but one of the many theories concerning the explanation of the clotting mechanism. There are many things yet to be found, and further investigation will complete this picture. Although recent discoveries have produced much progress, a great deal must be learned before the entire problem is solved.

CONDITIONS PREDISPOSING TO HEMORRHAGE AND THEIR TREATMENT

Accidental and operative trauma produce injury to blood vessels and bone, giving rise to hemorrhage. In accidental trauma one has little control of the situation, but in surgery trauma is definitely under the control of the operator. Thoma[8] states that trauma is the greatest single cause of hemorrhage. Surgical trauma can be reduced by handling soft and hard tissue with the greatest of care. The tearing of tissue should be avoided. Tissue should be severed from its attachment by sharp dissection. Blunt dissection may be used when tissue is being separated, but only to a point at which the tissue is not likely to be torn. Excessive tension must be avoided to prevent damage to tissue, blood vessels, and nerves.

Bleeding in bone may be controlled by pressure or by packing with gauze saturated in tannic acid solution.[9] If bleeding continues, then it is necessary to crush and condense the spongiosa with a burnisher. The use of bone paste is valuable, and the placing of bone wax can be helpful.

Irradiation, accidental or used as therapy to affected parts, produces injuries to blood vessels and the blood coagulation mechanism. Fatal hemorrhage may result from a minor surgical operation or injury to the irradiated parts. Griffiths and his associates found that rutin prevented increase in capillary fragility caused by intraperitoneal administration of radon ointment

in rats and in simulating the reparative processes following roentgen burns.[9] Clark, experimenting with guinea pigs, showed that rutin gives a high degree of protection against roentgen irradiation, sufficiently intense to cause widespread subcutaneous hemorrhage where vitamin C is ineffective.[10]

Patients treated with anticoagulants, such as heparin and Dicumarol, should have special attention when any type of surgery in emergency cases is considered. These drugs are used in the control of or prophylaxis against venous and carotid thrombosis or infarction. These medicaments are physiologic anticoagulants, preventing the proper agglutination of the platelets and inactivating thrombin. The effect of both of these medicaments can be controlled, counteracted, or inactivated with the use of toluidine blue or protamine.[11]

DISEASES PREDISPOSING TO HEMORRHAGE AND TREATMENT

Hemophilia.—Hemophilia is a congenital disease which is definitely hereditary and is sex linked in its transmission. It occurs only in the male and is transmitted directly and solely by the female. Although the female transmits the disease, she is symptom-free. There is but one blood finding characteristic of hemophilia—the prolongation of the coagulation time, while the bleeding time remains normal.[12] Clinically, hemophilia is characterized by excessive hemorrhage, which may be spontaneous or may follow injury. It assumes a serious aspect and becomes a problem of major importance because it may be complicated by severe and uncontrollable hemorrhage. The etiology of hemophilia is a mystery; some claim that it is due to a lack of ovarian hormone.[12] The bleeding may occur from any place in the body, under the skin, into the muscles, or from the nose, mouth, stomach, or intestines.

In the treatment of the hemophiliac, the family and past histories are important. The coagulation time must be reduced to normal before surgery is contemplated for this type of patient. It has been found that tissue thromboplastin or normal platelet-free plasma added to hemophiliac blood restores the normal clotting time. The missing thromboplastic material may be furnished either by blood or by the application of tissue extract to the bleeding point.[10] The coagulation time must be normal at the time of operation and should be kept at near normal levels for a period of four days or longer following the operation. Lewis and Jack[13] state that placental extract is the best means for temporarily lowering the coagulation time as a preventive measure. Locally, the application of tissue extract, such as thrombin, with pressure aids in the control of hemorrhage.

For the best care of a hemophiliac, there must be close cooperation between the oral surgeon and the hematologist.

Pseudohemophilia.—Pseudohemophilia is a hereditary disease occurring in both sexes and transmitted by both parents. The clot which forms is faulty, so that hemorrhage in the usual physiologic manner is not arrested. Although

the platelet count is within normal limits, this condition is produced by improper agglutination of the platelets. Pseudohemophilia is not so serious as hemophilia or severe purpura, but patients with the disease do require careful treatment.

Anemia.—In the anemias there is excessive reduction of erythrocytes, hemoglobin, or both. In pernicious anemia, the erythrocytes may be reduced below 2,000,000 with signs of purpura and prolonged bleeding following the extraction of a single tooth. Aplastic anemia may follow exposure to certain physical agents and chemicals. It has a very rapid onset predisposing to prolonged hemorrhage.

Before surgery is undertaken in an anemic patient, the condition must be corrected. Adequate diet and vitamin administration should be included with specific treatment for the type of anemia present. Folic acid, vitamin B_{12}, and liver concentrates are used to treat pernicious anemia or macrocytic anemia of the pernicious anemia type with a megaloblastic bone marrow. For anemias due to iron deficiency, whole liver is used to furnish iron and protein. Certain megaloblastic anemias fail to respond to vitamin B_{12} but react to folic acid. If surgery is necessary, whole blood should be administered during surgery and postoperatively to maintain a normal red cell count and hemoglobin.[10]

Leukemia.—Leukemia is a disease characterized by a widespread increased production of leukocytes, many of which are not mature. This condition is the most widely encountered blood dyscrasia and is considered a malignant neoplastic disease.[14] It occurs in acute and chronic forms of myelogenous, lymphatic, and monocytic leukemia. It may have definite oral symptoms predisposing to prolonged bleeding, and all forms are fatal.

In leukemia the diagnosis is confirmed by a complete blood count and marrow smear. With the slightest trauma, excessive bleeding will occur. After positive confirmation of the disease, only the essential procedures should be performed. Blood transfusion is the best treatment, and the use of thrombin-soaked Gelfoam locally helps control bleeding. When teeth are being removed, splints may be helpful in the placement of Gelfoam.

Purpura.—The term *purpura* is applied to various hemorrhagic diseases in which spontaneous bleeding occurs beneath the skin, from mucous membranes, or into joints. The subcutaneous hemorrhages appear as small or large purplish spots which gradually pass through the color changes characteristic of a bruise. Since purpura is so varied in its characteristics and occurs in diseased states that are so widely different, it should be considered as a symptom of a disorder of the blood vascular system rather than as a disease in itself. This condition has the outstanding feature of an enormous decrease in the number of blood platelets. Purpura may be produced by the depression of bone marrow actively by drugs, replacement of bone marrow by foreign tissue, or even a deficiency of nutritional factors due to anemia or scurvy.[5] The coagulation time is usually normal, with a soft clot which will not retract, but the bleeding time is prolonged.

There has been no accepted etiology for the purpural diseases. Usually a splenectomy produces an increase in the number of blood platelets and frequently results in a cure. Best and Taylor[10] report that this condition has been successfully treated with snake venom.

Hypoprothrombinemia.—Hypoprothrombinemia may be caused by various conditions which inhibit or interfere with the growth of normal intestinal flora, preventing the uptake and utilization of vitamin K.[9] It also occurs with severe liver damage. A bleeding tendency is not shown until the prothrombin concentration falls to nearly 30 per cent of the normal. The hypoprothrombinemia may be a result of presurgical sterilization of the gut interfering with vitamin K production. A deficiency of bile in the intestine produced by obstructive jaundice will interfere with the vitamin K, since bile is necessary for vitamin K absorption. The administration of vitamin K will compensate this type of insufficient synthesis. Prothrombin synthesis will be interfered with, if a normal liver function is not maintained. Some types of hypoprothrombinemia may be fully corrected only by restoring normal liver function, but with serious liver disease this is often impossible.

Polycythemia.—Polycythemia is not secondary to any known pathologic condition. It is a disease in which the blood cell count is above 7,000,000.[15] The red bone marrow is greatly increased in amount and is very hyperplastic. The viscosity of the blood is elevated, predisposing the patient to thrombosis formation. Patients suffering from this disease are potential surgery risks due to the increased red cell count and a tendency to bleed.

For the treatment of polycythemia, the high red cell count can be reduced by feeding whole beef or hog liver or by daily injections of ascorbic acid. The effect of liver suggests the presence of an hepatic hormone possessing a depressant action upon bone marrow activity. Radioactive phosphorus[32] has been used with success lately in the treatment of the disease.[10]

Hypothrombinemia.—In hypothrombinemia there is a reduction in the level of prothrombin plasma in chronic liver disease. This reduction of prothrombin may be as low as one-half or lower than the normal value. It gives rise to bleeding conditions. Prolonged salicylate therapy also leads to abnormal clotting in hypothrombinemia.[16] Prothrombin is produced in the liver with the aid of vitamin K. If there is any liver disease present, or if there is a decrease in vitamin K from the intestinal flora, hypothrombinemia will result. These conditions must return to normal before any surgery is undertaken.

Jaundice.—In hemolytic, infectious, or obstructive jaundice there is an inhibition of vitamin K absorption in the intestine as well as prothrombin formation in the liver.[15] Because of this condition, there is a lack of prothrombin, predisposing the patient to postoperative hemorrhage. This prohibits the formation of fibrin, and the clotting mechanism is impaired.

In obstructive jaundice or liver damage, the liver may not be able to supply sufficient prothrombin to form an effective clot. In preparing the

patient for surgery, it is possible to restore the prothrombin with the administration of vitamin K. When the prothrombin time is reduced to 20 per cent of normal, bleeding will occur.[17]

Scurvy.—The disease known as scurvy or hypovitaminosis C is caused by a deficiency of vitamin C and is rarely seen today. The oral symptoms present definite manifestations, with inflammatory, spongy, bleeding gingivae leading to prolonged hemorrhage in surgery. Ecchymosis may be present on the palate, and hematoma formation may lead to ulceration of the gingiva.

Scurvy causes abnormal fragility of the capillary walls. This condition demonstrates that a dietary deficiency exists due to the lack of vitamins C and P, which are contained in fruits and vegetables. Scurvy responds to the administration of increased amounts of vegetables, citrus fruit, and vitamins C and P. Proper oral hygiene and improved care of the local condition helps to shorten the recovery period.[18] Rutin acts on capillaries in much the same way as vitamin P.

Arteriosclerosis.—Prolonged bleeding may occur in arteriosclerosis, especially when the latter is associated with hypertension. Because of the arteriosclerosis, the capillaries are less contractile, and there is increased blood pressure due to hypertension. These two types of disease must be brought under control before surgery is performed. The patient is usually aware of his condition because he is under the care of his physician. High blood pressure tends to encourage dislodgment of the clot. In caring for these patients, it is best not to extract teeth over too great an area at any one time. Careful attention should be given to the suturing of all bleeding points during the closing of the tissue.

Infection.—The presence of infection can lead to postoperative hemorrhage. Infection must be controlled to reduce the possibility of abscess formation. An abscess could possibly lead to erosion of tissue involving a major blood vessel, with an eventual rupture of the vessel. An active case of Vincent's infection in oral operations can produce postoperative hemorrhage. Under this condition, spontaneous bleeding from blood vessels ruptured by infection is very difficult to control.[19]

Diabetes.—Diabetes is a condition which produces postoperative hemorrhage because there is usually a tendency toward hyperemia of the gingival tissue. Persistent oozing may occur from the tissue after surgery has been performed, and diabetic patients have less resistance to infection. Diabetes leads to superficial infection around the margins of the sockets, with secondary hemorrhage resulting. The diabetic usually suffers from some vascular damage which, in itself, may lead to some local hemorrhage. The bleeding here is not serious, but care must be taken while the tissue is being closed.

CLINICAL RECOGNITION OF HEMORRHAGE

A careful appraisal of each patient will eliminate much of the need for hematologic consultation. It is very important to obtain a complete history

in order to evaluate the patient's potential for postoperative hemorrhage. The oral surgeon must be able to recognize the clinical signs of hemorrhagic diseases. Drawing conclusions and inferences as to the operative fitness of the patient is a faculty which increases or is developed with experience.

The physical appearance of the patient will often give some indication of generalized disease.

The patient may be under the care of his physician. By simple questioning, it can be learned whether or not he is receiving medication for any systemic disease which could have a bearing on the course of treatment.

Knowing the patient's occupation may be of some value. There may be disturbances in the normal mechanism involved in hematosis among persons working in industries concerned with the use of chemicals, plastics, and radioisotopes.

A careful family history should be obtained from patients who are subject to bleeding.[20] The history should extend as far back in the family tree as possible, including the immediate family and all relatives. Special attention must be paid to the sex of each patient, since hemophilia is a disease of the male, while idiopathic thrombocytopenic purpura occurs most often in the female. The family history should include a review of any systemic diseases to which the relatives have been subject, particularly those in which bleeding has been a factor.

The physical examination is the next important step in the diagnosis or ruling-out of a hemorrhagic disease.

The data accumulated from the history, physical examination, physical appearance, and careful interrogation of the patient will indicate whether the patient exhibits any hemorrhagic tendencies. If any suspicion of bleeding is present, the oral surgeon should seek the aid of a hematologist to evaluate the risk.

The screening tests which the hematologist can perform are the determination of the bleeding and clotting times and the capillary-resistance test. With the results of these preliminary clinical procedures and information from the physical examination and history, the hematologist decides whether or not further determinations and confirmatory procedures should be performed.

BLOOD LOSS IN ORAL SURGERY

The blood loss of a vascular bed during a surgical operation depends upon the size and number of vessels surgically interrupted and the length of time that bleeding or oozing is allowed to continue. The effect of hemorrhage on the host depends upon a number of factors, especially the age and general health of the patient and the proportion of blood lost in relation to the patient's normal blood volume.

Johnson's[2] study of blood loss produced sufficient evidence that the hemorrhage occurring in oral surgery compares with the blood losses incurred in

major surgery. Johnson evaluated blood losses in oral surgery in a series of 175 patients who were given oral surgical treatment by eight oral surgeons and arrived at the following conclusions:[2]

1. Blood loss in the total group ranged from 35 to 912 ml., the average loss being 233 ml. When the duration of the operation is compared with blood loss in the group with fewer than sixteen extractions, 89 per cent operated upon in less than thirty minutes had a blood loss below 300 ml. If the duration of surgery surpassed thirty minutes, 57.2 per cent remained below the 300 ml. level, whereas 42.8 per cent sustained blood losses ranging from 300 to 500 ml.

2. In the group of patients who lost sixteen or more teeth in the operative procedure, 44.5 per cent had blood losses above 300 ml. When the operating time remained below thirty minutes in this group, 90.5 per cent did not hemorrhage in excess of the 300 ml. level. When the surgery exceeded thirty minutes in duration, however, 53.8 per cent of the patients sustained blood losses ranging from 300 to 912 ml. It was shown, both in the total hospital experience and in the data analyzed according to groups or oral surgeons, that time is the most important consideration in the evaluation of blood loss. The more prolonged the operating time, the greater the blood loss.

3. Time and blood loss are direct surgical antagonists. The longer the operating time, the greater the blood loss.

4. The blood losses in oral surgery may be predicted to some extent. While having some guide to prognosing blood loss is valuable in cases of severe blood loss, it can become an important aid to calculate such a loss as it occurs.

5. It was found that bleeding, clotting, and prothrombin times can be within normal limits in a person who hemorrhaged profusely during oral surgery. Conversely, positive results of these tests do not necessarily point to abnormal bleeding tendencies.

6. The most valuable guide in the prognosis of abnormal bleeding tendencies in the average dental patient was the history of previous episodes of excessive postextraction hemorrhage.

7. In general, if this study of 175 patients has any prognosticative value, it tends to show that when sixteen or more teeth are extracted with alveolotomy, nearly 50 per cent of the patients will sustain blood losses in excess of 300 ml.

8. This study has demonstrated that it is extremely important to be able to evaluate and forecast, with some degree of accuracy, the individual blood loss in order that replacement therapy may be instituted whenever needed to maintain physiologic fluid balance. The well-being of the patient is of primary importance, and the operative procedure elected by the oral surgeon is of secondary importance. With the criteria for actual losses recognized, the patient's condition will be safeguarded with adequate and early replacement therapy.

USE OF CALCIUM TO CONTROL HEMMORHAGE

The administration of calcium gluconate, viosterol, styptysate and ceanothyn by mouth does not reduce the coagulation time of normal patients by an amount great enough to be of any clinical value. Therefore, there is no justification for the continued clinical use of these agents in the hope of shortening coagulation time or reducing bleeding. The result of using these drugs can be disadvantageous to the patient. By relying on these valueless drugs, one runs the risk of depriving the patient of more adequate means to prevent or control hemorrhage.[21]

Replacement therapy of some kind is indicated, if only for the purpose of maintaining fluid levels within the tissue fluid compartments. The fluids of the tissue compartments enter the vascular compartments to maintain the blood volume when whole blood is lost. The amount of fluid entering the vascular system governs the resultant state of dehydration within the tissue compartments which is further affected by insensible fluid losses. To maintain the proper fluid level, intravenous administration of a solution of 5 or 10 per cent dextrose in water is indicated in those patients in whom anticipated blood losses will exceed 300 ml. Precautions governing administration is indicated when the discomfort of surgery in the oral cavity makes these patients extremely reluctant to accept fluids by mouth.

When hemorrhage is in excess of 500 ml., the administration of whole blood should be considered. The patient's physician should be consulted when a systemic condition is present. The complications resulting from promiscuous use and abuse of blood transfusion should be common knowledge to anyone employing this type of treatment.

When replacement therapy is deemed unnecessary, the postoperative therapeutic treatment should include a high-quality protein, vitamin supplement of B complex and C, and iron in the form of ferrous sulfate. Also, one must bear in mind the possibility of postoperative hemorrhage.

PREOPERATIVE AND POSTOPERATIVE MANAGEMENT OF EXTRACTION HEMORRHAGE[11]

The following means may be used in management of extraction hemorrhage:

A. *Local*

Pressure: Gauze packs, splints, pressure dressing, intra-alveolar packs, rubber bands[22]

Cautery (chemical): Ferric chloride, tannic acid, and the styptics

Cautery (electrical): Platinum wire cautery, high-frequency current

Ligature: Catgut or other absorbable suture for the ligation of bleeding points

Hemostats: Crushing "bleeders" at points of hemorrhage

Biologicals: Blood fractions (thrombin, etc.), drugs (Adrenalin), thromboplastin, fibrinogen, fibrin foam,[23] epinephrine, Cobefrin, extracts (snake venom), Adrenosem Salicylate[24]

Absorbable materials: Oxidized cellulose, gelatin sponge,[25] and calcium alginates[26]

B. *Systemic*

Whole blood, intravenously
Blood fractions (plasma,[27] Cohen's fractions I,[28] II, III, IV, V, VI)
Vitamin K parenterally or orally
Vitamin C parenterally or orally

SUMMARY

There is a constant need in oral surgery for therapeutic agents which will accelerate the coagulation of blood, so that bleeding will be diminished to a point where it is no longer troublesome.

It is important that the oral surgeon recognize the clinical signs of hemorrhagic diseases. Complete history, physical examination, and careful questioning will indicate whether the patient exhibits any hemorrhagic tendencies.

If any suspicion of bleeding is present, the oral surgeon should seek the aid of a physician or hematologist to evaluate the risk involved if a surgical procedure is contemplated. The patient's health is of primary importance, and the operative procedure under consideration is of secondary importance.

In the practice of oral surgery, the amount of hemorrhage has been underestimated. When replacement therapy is indicated, the hematologist should be consulted.

Any surgical procedure may give rise to a potential hemorrhage. The oral surgeon must anticipate and be able to treat postoperative hemorrhage.

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SURGICAL OPERATIONS ON THE ALVEOLAR RIDGE TO CORRECT OCCLUSAL ABNORMALITIES

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IN SPITE of excellent results obtained by orthodontists, lately there has been a noticeable increase in the number of patients, especially of post-adolescent age, seeking maxillofacial surgery for correction of pronounced deformities and malformations of the jaws and dental arches. Some of these malformations went unnoticed by the patients and their relatives, and treatment had been requested too late. Such deformities impair facial harmony, and after puberty they often produce psychic depression and inferiority complexes. These, in turn, may interfere with normal employment as the person loses self-confidence. The patients are more anxious to submit to treatment at this age than when they were younger, and they are even ready to undergo surgical intervention. The short time required for surgical correction of these anomalies further justifies these operations.

The choice between orthodontic and surgical treatment is based on the severity of the deformity and the age of the patient. Generally, surgery is to be preferred when the malformations are very pronounced or when bone growth has ceased. Such operations are justified also when the entire permanent dentition has been completely formed and only partially satisfactory results may be achieved by conservative orthodontic therapy.

In this article will be presented various surgical methods which may be performed on the alveolar process to correct these malformations of the jaws and malpositions of the teeth in lieu of complete osteotomy or osteectomy of the jaws. These interventions may be classified as follows:

1. Purely surgical procedures which completely replace orthodontic treatment.
2. Surgical procedures to facilitate subsequent orthodontic treatment.

PURELY SURGICAL PROCEDURES REPLACING ORTHODONTIC TREATMENT

The first group includes operations for mandibular alveolar retrusion and protrusion, maxillary protrusion, and open-bite. Treating these dysgnathic conditions by osteotomy of the alveolar ridge in the interdental spaces requires an extremely exact procedure. An error of 1 mm. may injure the alveolus of the neighboring teeth and devitalize them.

From Graz University Dental Clinic, Department of Maxillo-Facial Surgery (Prof. R. Trauner, Director).

Fig. 1.

Fig. 2.

Fig. 1.—Metal band splint.
Fig. 2.—Articulated splint.

A. *B.*

C. *D.*

Fig. 3.—Case with alveolomandibular retrusion. *A* and *C*, Photograph and roentgenogram of profile before operation. *B*, Photograph five months after operation. *D*, Roentgenogram seven weeks after the operation.

It is unjust to diagnose these anomalies only on the basis of the occlusal relationship. The correlation of the teeth and jaws to the face and the skull should always be taken into consideration. These relationships can be determined from teleroentgenographic films. A good profile snapshot and dental plaster casts can give valuable information. These operations should be performed first on models, since the adjustment of the sections may bring to light many important details.

The splinting is generally done with metal bands. The bands are first adjusted on each tooth in the mouth, and later they are soldered together in the laboratory where a metal bow with lugs is attached to the buccal side (Fig. 1).

Wire splinting, according to Stout,[39] Obwegeser,[24] and others, may be used only when there is a good interdental contact. An articulated splint, according to Kersting, is preferred when no intermaxillary fixation is used. It permits normal mouth functions (Fig. 2).

Anterior Alveolar Retrusion in Normal Mandible.—There is a rare form of mandibular retrusion in which the alveolar ridge with the lower teeth is placed distally as much as 8 to 12 mm. The lower lip is indrawn, the labiomental crease is pronounced, and yet the mandible and chin are developed normally (Fig. 3).

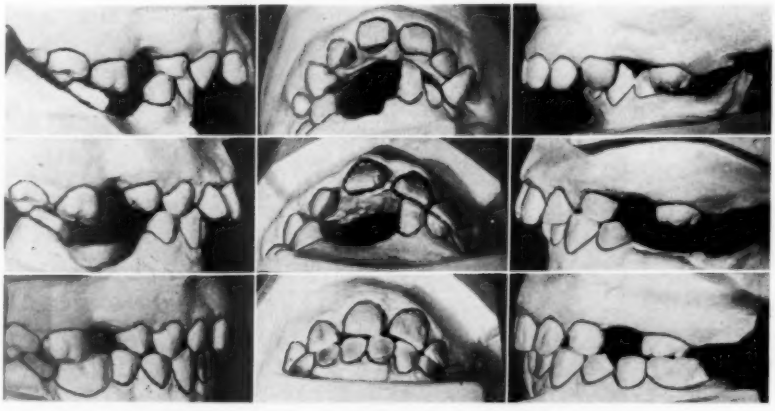


Fig. 4.—Plaster models of the case shown in Fig. 3. *A*, Before treatment. *B*, After operation on mandible. *C*, After prosthetic restoration.

This alveolomandibular retrusion is usually caused by the absence of $\overline{6,5}\,|\,\overline{5,6}$ or of $\overline{5}\,|\,\overline{5}$ or by the premature extraction of $\overline{6}\,|\,\overline{6}$. The lower front teeth have been wandering distally. The malformation is often associated with maxillary protrusion, caused by the pressure of the lower lip on the palatal surface of the upper incisors (Fig. 4). The deformity is extremely disfiguring. An operation to eliminate and correct this anomaly has been

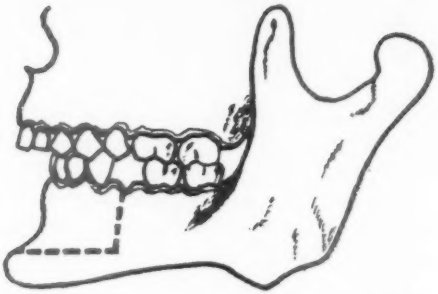
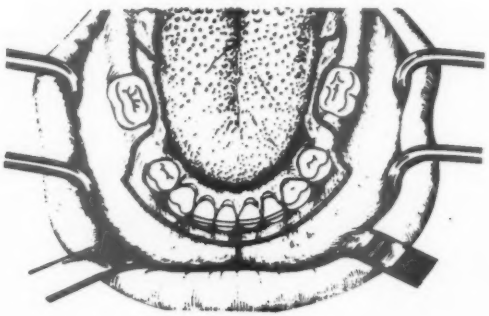


Fig. 5.—Hofer's method of advancing the entire alveolar fragment in case of alveolomandibular retrusion.

Fig. 6.



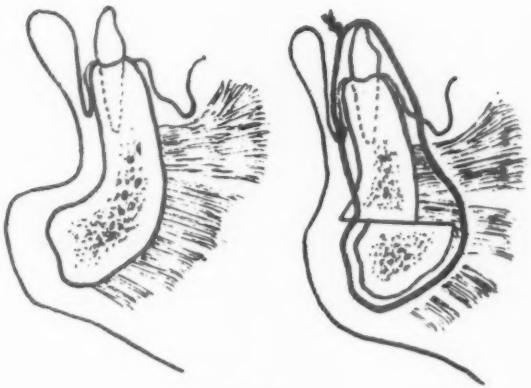


Fig. 7.

Fig. 6.—New mucosal incision (Köle).
Fig. 7.—Cross section showing osteotomy and circumferential wiring.

described by Hofer,[12, 13] who proposed that the entire alveolar ridge, including the teeth, be advanced (Fig. 5). The operation is performed first on plaster models in order to achieve a favorable occlusion and to prepare the articulated splint used in this procedure. Hofer's mucosal incision is made on the buccal gingival crest with a vertical extension on each side. It also severs the mental nerve. This incision allows only the tilting of the alveolar ridge, however, thus protruding the teeth. It does not advance the osteotomized fragment.

I prefer to make the incision in the vestibule, circumventing the mental nerve and leaving the entire mobilized fragment covered with mucosa. The incision is then carried over the alveolar ridge and along the lingual gingival margin to the retromandibular triangle, with a vertical extension medianward (Fig. 6). This extensive incision permits the elongation of the mobilized mucosa and allows the protrusion of the osteotomized bone, and not merely its tilting. Holes are drilled with a round burr, approximately 1 cm. below the apices of the teeth, and joined by means of a fissure burr. Now the fragment is mobilized by chiseling, first with fine, sharp osteotomes followed by larger ones. The detached fragment may now be easily advanced by as much as 2 cm. if necessary. It is fixed in position by a splint. The vertical fixation is achieved by a circumferential wire passing through a previously prepared hole in the median and foremost point of the fragment and twisted over the splint (Figs. 7 and 8). This fixation counteracts the pull by the musculature in the floor of the mouth, which tends to draw the fragment backward. The splint and circumferential wire are removed approximately six weeks after the intervention, when sufficient bone consolidation has taken place.

Fig. 8.—Photograph taken during the operation, showing osteotomy and circumferential wiring.

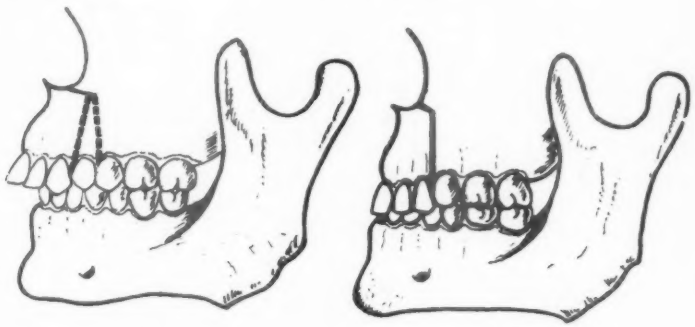
It is of great importance in this operation to cut the alveolar segment well below the apices of the teeth to prevent injuring them. The bone section at this point is larger, and it may therefore be carried forward a considerable distance. The large surface also favors a better consolidation, and the nutritive pedicle is formed not only by the mucosa but by the genioglossus and geniohyoid muscles as well. The mental nerve, though not severed, suffers

light traumatization, and lower lip anesthesia lasting two or three months may result. The temporarily negative result of the vitality test of the lower teeth disappears completely in six months. After removal of the fixation, the gaps should be maintained by bridgework or a prosthetic appliance.

The surgical correction of these rare anomalies gives very satisfactory results. The facial deformity is corrected, and the occlusion attained permits a marked functional improvement.

Maxillary Protrusion.—Maxillary protrusion is by far the most frequent anomaly. It is only by surgical intervention that it is possible to correct the protrusion of the teeth and improve the short upper lip. Difficulties with mastication and speech impediments are other indications for surgical intervention.

The operation to correct maxillary protrusion is best performed on patients between the ages of 16 and 30 years. At this age it is either too late for orthodontic treatment or a relapse is probable.



A. *B.*

Fig. 9.—Wassmund's technique for retrusion of the maxillary median fragment, thus causing a deep bite.

There are two forms of maxillary protrusion—one with crowded teeth and the other with diastemas between the teeth. We shall take up the second type later. The principles of the operation for maxillary protrusion with crowded teeth were given by Wassmund[38] and Spanier.[31] A premolar is extracted on each side to obtain a space for the retrusion of the maxillary median fragment. The alveolar bone is removed buccally and palatally. Horizontal osteotomy is performed over the apices to the anterior bony aperture of the nose and on the hard palate. Schuchardt[27-30] uses the same principle but operates in two stages. When performing the horizontal osteotomy, he insists on removing bone above the teeth as well as cartilage from the nasal septum. The postoperative splinting is done either by a palatal bow[38] or by a labial bow.[27-30] In this operation the maxillary fragment is tilted downward and lingually. The deeper the bite and the more distally retruded the fragment, the deeper will be the overbite (Fig. 9).

Because the esthetic as well as the functional results of this deep overbite are rather poor, especially if the lip is short, bite plates are attached to the lateral teeth in order to prevent the overbite. Once they are removed, an open-bite results in the premolar and molar region. As the posterior teeth do not grow fast enough to close the open-bite, the maxillary teeth are pushed forward by the lower incisors. Even though bone consolidation has reached a satisfactory condition, this pressure produces a relapse. To prevent a relapse, prolonged orthodontic treatment for a period of one to two years is necessary.[1, 2, 15, 16, 27-30] This is the major reason so many surgeons shrink away from this operation. I move the splinted fragment postoperatively into the desired position with the help of rubber band traction of the type used in treating fractures of the jaw. This is a timesaving procedure for both the patient and the surgeon.

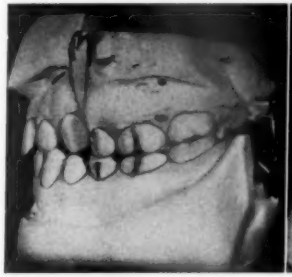


Fig. 10.

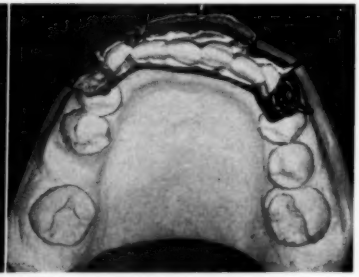


Fig. 11.

Fig. 10.—Step on the upper surface of the model.
Fig. 11.—Cast splint for vertical displacement of the mobilized maxillary fragment.

First, the operation is performed on plaster models. These models sometimes show that slight grinding of the incisal edge of the lower teeth is necessary (Fig. 10). The model study also permits the exact evaluation of the bone to be ostectomized for the distal (approximately 5 to 9 mm.) as well as the vertical (approximately 4 to 7 mm.) displacement of the fragment. The exact amount of bone for the vertical and horizontal ostectomy is determined from and marked on the models. Metal band arch splints are now adjusted for the front and lateral teeth (except for the teeth to be extracted) as well as for the lower lateral teeth. A cast splint is cemented on the lower front teeth. The cast splint has a rest on which the upper incisors rest, elevating the fragment into the desired position (Fig. 11). Lugs are soldered to these splints for the attachment of elastic bands. A vertical mucoperiosteal incision is made on the vestibular side, one tooth's width behind the tooth to be removed. The bone is exposed and the tooth is extracted. Two vertical cuts in the bone are made, corresponding in width to the desired distal displacement. The bone excision, corresponding in width to the vertical displacement, is then carried on horizontally 1 cm. above the apices to the lateral nasal aperture. It is of utmost importance to remove the bone horizontally

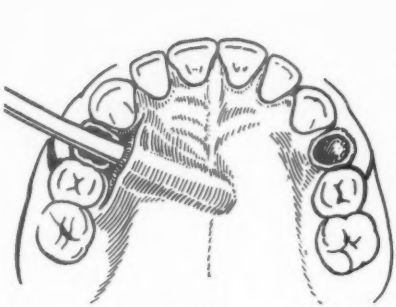


Fig. 12.

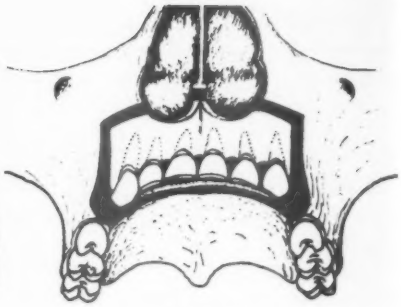
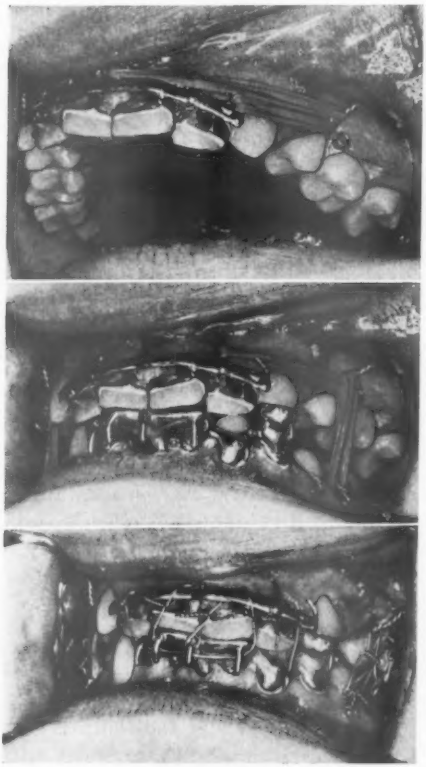


Fig. 13.

Fig. 12.—Undermining of the palatal mucoperiosteum.
Fig. 13.—Sketch of the complete operation.



A.

B.

C.

Fig. 14.—Photographs showing the posterior and vertical repositioning in the mouth. *A*, Elastics are applied horizontally. *B*, The deep bite is avoided by splints, causing a posterior open-bite. Vertical elastics are applied to correct the open-bite. *C*, The maxillary median fragment is elevated; thus, normal occlusion is obtained posteriorly. Finally, the elastics are replaced by intermaxillary wires.

to the exact extent shown on the model, as otherwise the vertical displacement of the fragment will be hindered. The palatal mucoperiosteum is only undermined[8] and the alveolar bone removed (Fig. 12). From the top of this alveolus where the tooth has been extracted, a single bone incision is carried medially and is joined to a similar incision made on the other side of the

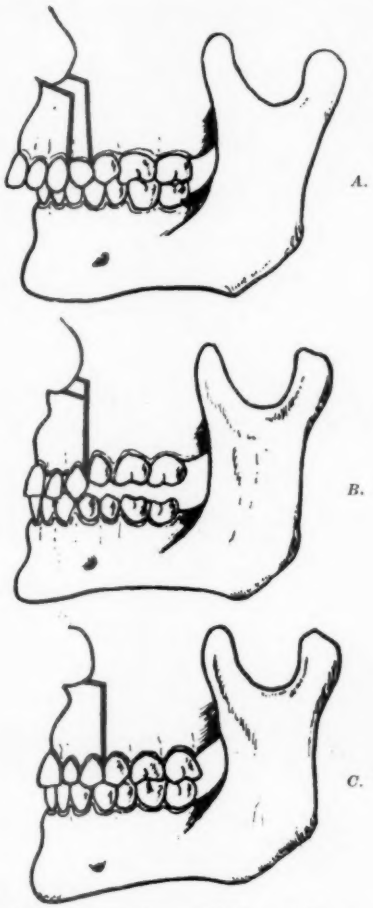


Fig. 15.—Sketch of the posterior and vertical repositioning of the maxillary median fragment.

maxilla. Now the bone strips are removed, and the fragment is totally mobilized by chiseling with fine osteotomes. Next, an internasal incision on the anterior border of the septum is made. The cartilaginous septum is detached from both its mucosal and bone attachments. A horizontal strip from this cartilage, corresponding in height to the vertical displacement of the fragment, is removed. Then all incisions are sutured (Fig. 13).

The nutritive pedicle for the mobilized fragment of the maxilla consists of the mucoperiosteum between the two cuspids in the vestibule and the palate. The repositioning of the fragment starts five days later (Fig. 15, *A*).

Fig. 16.—Case of maxillary protrusion. *A* and *C*, Before the operation. *B* and *D*, After the operation.

First, elastics are applied horizontally between the lugs on the splints of the medial fragment and those of the lateral fragment (Fig. 14, *A*) until contact is obtained between the teeth on each side of the ostectomies (Figs. 14, *A* and 15, *A*). The upper incisors rest now on the bite plate of the lower splint, causing an open-bite posteriorly. To eliminate this, elastic traction is applied between the upper and lower posterior splints (Fig. 14, *B*). This forces the median fragment whose teeth occlude on the lower median splint to become elevated vertically (Figs. 14, *C* and 15, *C*). Once the desired result has been

achieved, which may be in one or two days, the elastics are replaced by intermaxillary wires. This mode of fixation is maintained for three weeks and presents the only disadvantage of my method. After the intermaxillary wiring and the splints are removed, the retention of the position of the fragment is

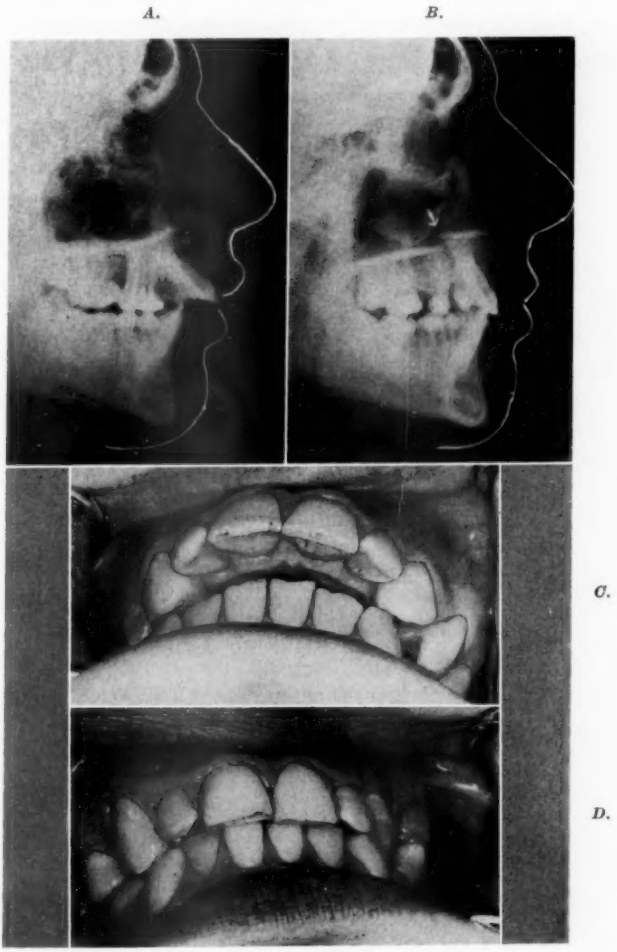


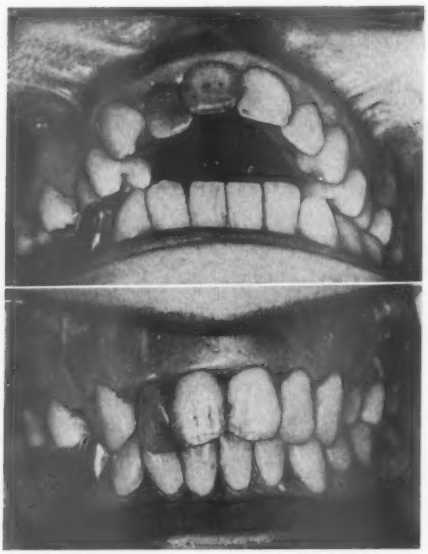
Fig. 17.—Case shown in Fig. 16. Roentgenograms taken before (*A*) and after (*B*) operation. Preoperative occlusion (*C*) and postoperative occlusion (*D*).

assured for the next three weeks, until sufficient bone consolidation has been achieved, by a palatal plate tied with silk to the teeth. The entire procedure —operation and treatment—is finished within six weeks (Figs. 16 and 17).

If the maxillary protrusion is combined with an open-bite, as in tongue-biting, we may dispense with the lower median splint without fear of any

cosmetic or functional disadvantages (Fig. 18). Special care should be taken in cases in which maxillary protrusion exists simultaneously with distoclusion. In such cases, one should consider whether or not a better cosmetic result could be obtained by protruding the mandible. In the usual case of distoclusion, however, a better result is certainly obtained with either the retroauricular cartilage implantation[33-37] or step-shaped vertical ramus osteotomy.[24]

A.



B.

Fig. 18.—Open-bite case (tongue-biting) before (*A*) and after (*B*) operation.

Both of these methods may be used to protrude the mandible. Sometimes it is advisable to combine the correction of maxillary protrusion with an operation for enlargement of the chin.

(This article will be continued in forthcoming issues of the Journal. References for the entire article will appear at the end of the article.)

SQUAMOUS-CELL CARCINOMA ARISING IN MUCOSA OVERLYING TORUS PALATINUS

Report of a Case

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AN EXOSTOSIS occurring in the midline of the palate is termed torus palatinus, whereas one located unilaterally or bilaterally on the lingual aspect of the mandible between the cuspid and the molars is called torus mandibularis. These tori vary greatly in shape and size. Torus palatinus may be flat, nodular, spindle shaped, or lobular. The etiology of these tori is not fully known. Among the accepted views of etiology are the functional response, sex, race, and heredity. Kolas and co-workers,[1] in their extensive study of 2,478 dental patients, have concluded that palatine tori occurred in 20.9 per cent and mandibular tori in 7.75 per cent of the total group. Sex distribution for torus palatinus revealed a significant difference in percentage (males 14.7 per cent and females 26.7 per cent) with approximately a 2:1 ratio in favor of females. There was no significant racial difference. Kolas and his colleagues suggested that the onset of these tori may be found up to the third decade. Miller and Roth,[2] in reporting a similar study, pointed out that torus palatinus before the age of 5 is rare and that this condition first appears as a slight torus at about the age of 7. The middle 50 per cent of the cases of torus palatinus are between the ages of 20.8 and 50.2 years, with an average age of 35.9 years. These authors agree with Kolas and co-workers[1] that the ratio of torus palatinus is 2:1 in favor of females. They have also pointed out that torus palatinus grows rather gradually, taking an average of 8.4 years to grow from a slight to a moderate size.

Torus palatinus is a benign growth and usually is of little significance. Occasionally, the patient with such a lesion may experience some impairment of speech, or there may be difficulty in the construction of artificial dentures. The patient, knowing of the presence of this tumor, may be worried about cancer. The following case is being reported because of its unusual nature, in that a squamous-cell carcinoma developed in mucosa overlying an already existing torus palatinus.

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CASE REPORT

A 57-year-old white woman was referred to M. D. Anderson Hospital on Sept. 12, 1956. At that time her chief complaint was an ulcer in the roof of her mouth. She had been to her own physician, who had referred her to an oral surgeon. A biopsy had been taken and the pathologist's report indicated a squamous-cell carcinoma, grade 1.

Fig. 1.—Torus palatinus, the right lateral anterior surface of which was ulcerated and had a heaped-up border. Biopsy report was positive for squamous-cell carcinoma, grade 1.

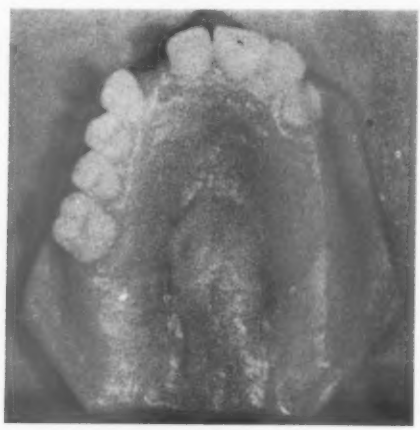

Fig. 2.—Stone cast showing the lesion.

On examination, the patient stated that she had had torus palatinus for many years. This tumor was oval in shape and measured about 3 cm. in the greatest diameter. The right lateral anterior surface presented an area of 3 to 4 mm. superficial ulceration with a raised, heaped-up border. The greatest diameter of the lesion was about 1 cm. This was the area from which the previous biopsy had been taken and reported as squamous-cell carcinoma (Figs. 1 and 2).

Past History.—The patient had had an appendectomy and hysterectomy in 1932, a tonsillectomy between 1935 and 1940, a cholecystectomy in 1946, and a hemorrhoidectomy in 1954. A lipoma and a sebaceous cyst had been removed from the right breast and the left side of the chest.

She had sustained bilateral fractures of the clavicle, in both arms, and both wrists in childhood.

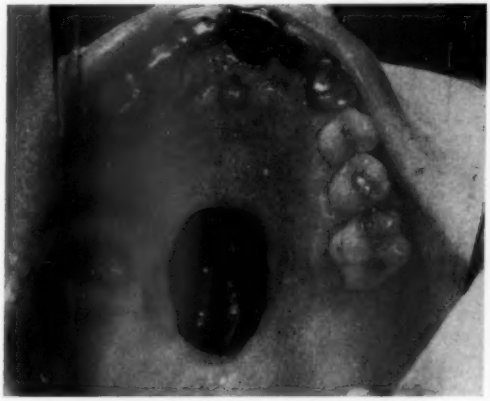

Fig. 3.—Cleft in the palate (postoperative).

Fig. 4.—Stone cast of cleft palate.

Medical History.—The patient denied having had any childhood disease. She had had typhoid fever at 1½ years of age and thrombophlebitis about ten years before admission. An embedded kidney stone in the right kidney had been diagnosed five years before she came to Anderson Hospital.

Personal and family history revealed nothing remarkable.

Treatment and Operative Procedure.—The patient was operated upon on Sept. 20, 1956, under general endotracheal anesthesia. By means of electric cautery, an incision was made

through the mucous membrane overlying the hard palate. This incision was carried along the posterior margin of the overlying torus palatinus and along the lateral and anterior borders approximately 1.5 cm. from the gingival margin. After the incision was carried down to the bone, the hard palate was divided along the lateral margin and the vomer was divided, freeing the hard palate, by means of a hammer and chisel. The tumor with the attached portion of bone was removed. The inferior turbinates were also removed. The raw surfaces of the wound of the hard palate were cauterized. Bleeding was controlled with gauze packing soaked in neomycin. A previously devised obturator was inserted immediately, thus obviating the use of a feeding tube. The patient received a transfusion of 500 c.c. of whole blood during the operative procedure. The postoperative course was uneventful, and she was discharged on the eighth postoperative day.

Fig. 5.—Prosthetic appliance (permanent obturator).

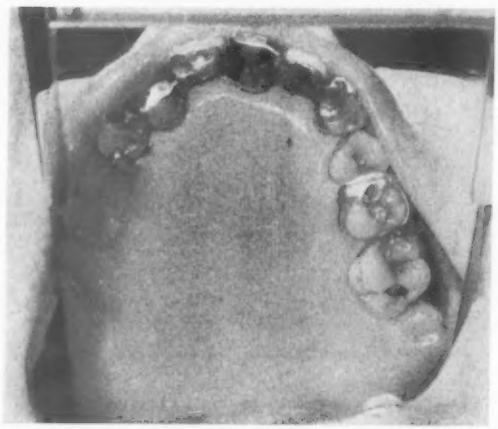

Fig. 6.—Obturator in position.

Routine follow-up has been carried out without evidence of recurrence. The patient was last seen on Dec. 30, 1957. The immediate temporary obturator has been replaced by a permanent obturator (Figs. 3 to 6).

SUMMARY

A case of squamous-cell carcinoma in the mucous membrane overlying a torus palatinus is reported. Although it is unusual for these tori to take a cancerous course, any long-persisting ulcers in or around such benign ostoses should be considered potentially malignant.

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6516 JOHN FREEMAN AVE.

LYMPHOSARCOMA OF THE MAXILLA AND MANDIBLE

Report of a Case

Raymond J. Englert, D.D.S., and Harry N. Pasqual, B.S., M. Litt., D.D.S., Pittsburgh, Pa.

LYMPHOSARCOMA is a tumor which arises in one group of lymph nodes or in one collection of lymphoid tissue and spreads to other groups of nodes, apparently by way of the lymphatics.[1]

This highly malignant, rapidly growing tumor is characterized by its tendency to destroy the capsule of the nodes, by its proliferative growth, by the invasion of surrounding tissues and bone, and by its extreme radiosensitivity with rapid recession of the tumor mass following radiation. Metastases are common, and the prognosis is very unfavorable.

Rollins and Thoma[2] have reported a case of a solitary lesion of the mandible that responded very well to radiation therapy.

CASE REPORT

History.—W. T., a 19-year-old white boy, presented a history of numbness, swelling, and drainage from the gingival tissue around the right mandibular third molar area. These symptoms had been present for the past month. He visited his local dentist, who examined the area and extracted the right mandibular third molar. The drainage and swelling persisted, and the patient noted an increase in the size of the "abscess" with some soreness present. He visited another dentist, who became suspicious of the area and referred the patient for specialized treatment. A biopsy of the tissue was made and the patient was admitted to the St. Francis General Hospital in Pittsburgh, Pennsylvania, on June 26, 1957.

On admission, the patient stated that in addition to the above symptoms he had noted some vague abdominal pain during the past couple of months. This was described as a pressure feeling in the lower abdomen. He had suffered a loss of appetite together with a weight loss of approximately 20 pounds in the past month. Bowel movements were normal.

Oral examination revealed a necrotic mass, measuring approximately 4 by 3 cm., in the right mandibular molar area. The mass was firm and indurated, and it projected below the angle of the mandible. The upper right and lower left gingival tissues were edematous and red (Figs. 1 and 2). The masses were firm but not especially tender. Slight cervical adenopathy was noted.

Physical examination revealed a large suprapubic, firm globular type of mass, approximately 10 cm. in diameter, just below the umbilicus. Blood pressure was 128/84, pulse was 88, and respirations were 20 per minute. The patient was in no acute distress.

On the following day a complete gastrointestinal study was carried out and reported as negative. A skeletal x-ray survey was negative. Examination of the chest revealed some pleural effusion. A bone marrow examination revealed very cellular marrow with no abnormal cells. About 50 per cent of the myeloblasts showed vacuoles in the cytoplasm, the nucleus, or both. This was reported as being seen frequently in leukemia, but no other findings of leukemia were present in this case.

A second biopsy specimen was obtained from the mass in the right mandibular third molar area and was sent to the laboratory for routine pathological examination.

*Histopathologic Report.**—Macroscopically, the specimen consisted of a piece of tissue from the oral cavity which measured 2.0 by 1.7 by 0.6 cm. The tissue was received in Zenker's solution and was discolored yellowish tan. Microscopically, the sections of tissue were partially covered on one surface by a thick layer of stratified squamous epithelium.

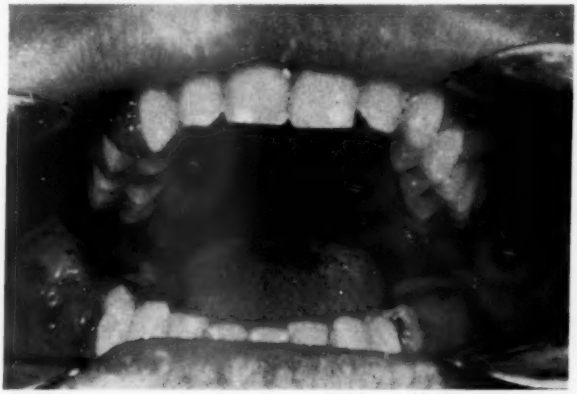

Fig. 1.—Lymphosarcoma of the maxillary and mandibular gingival tissues. Bilateral involvement.

Fig. 2.—Note the proliferative growth on the buccal and lingual tissues.

The underlying edematous stroma contained large masses of very deep-staining small cells which had markedly vesicular nuclei and no identifiable cytoplasm. These cells varied only slightly in size and shape and formed no definite structural pattern (Figs. 3 and 4).

Pathologic diagnosis: Lymphosarcoma of the gingiva.

The patient was examined at the tumor clinic and was considered inoperable. He was placed on urethane and Thorazine and also received a series of x-ray treatments to the

*The pathologic reports were prepared by Dr. Robert C. Hamilton, Director, Department of Pathology, St. Francis General Hospital.

Fig. 3.

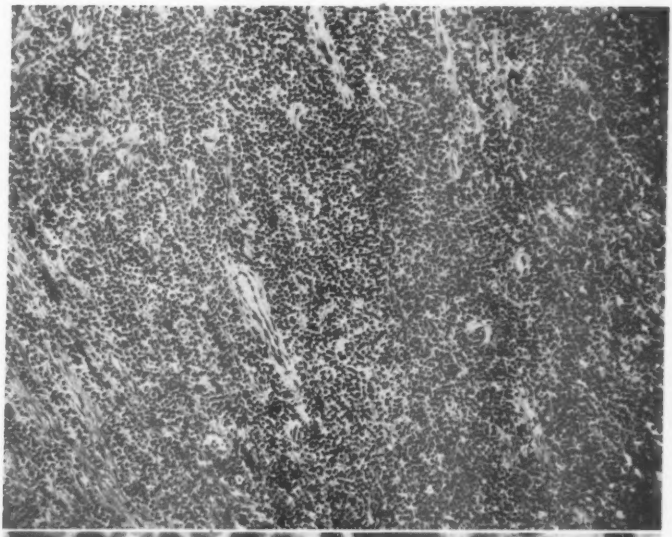

Fig. 4.

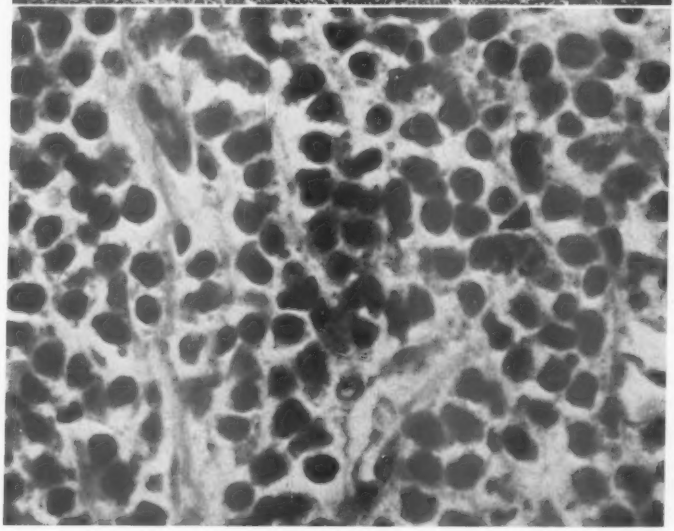

Fig. 3.—Low-power section of lymphosarcoma.
Fig. 4.—High-power photomicrograph with many mitotic figures present.

maxilla, mandible, and abdomen. There was a rapid recession of the evident local tumors of the oral cavity and abdomen, followed by an extensive spread to both pleural cavities. The patient died twenty-three days following his admission.

DISCUSSION

Lymphosarcoma appears to favor no particular age group or sex. It is a very destructible neoplasm, even though it is known to be extremely radiosensitive. This case demonstrates its insidious onset and quick termination. Although it offers a poor prognosis, it is still imperative that an early diagnosis be established. Surgery and radiation may sustain the patient or provide a temporary cure.

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510 MEDICAL ARTS BLDG.

EXODONTIA

REMOVAL OF LINGUAL APEX UTILIZING PALATAL APPROACH

Marvin Friedman, D.D.S., Brooklyn, N. Y.*

FRACTURE of the lingual root of an upper molar is not an uncommon occurrence during exodontic procedures. When the fracture leaves the root level with the alveolar process, removal by means of elevators is usually successful. However, if the fracture leaves the remaining root deeply buried within the alveolar process and not visible to the naked eye, a flap type of operation is usually indicated for optimal access and visibility. The employment of an open procedure reduces the chance of accidental displacement into the maxillary sinus and usually results in shortened operating time, less trauma, and quicker healing.

The most common approach to this problem calls for creation of a buccal flap, removal of the buccal plate and interseptal bone to bring the lingual root into view, and removal of the root by means of suitable elevators. This technique is not entirely satisfactory, for it involves breaking down a substantial portion of the buccal alveolus as well as interseptal bone in order to visualize the apex and establish access. This is a time-consuming procedure, and when it is completed the operator is still working at a distance from the root surface. The shape of the alveolar ridge has also been altered in an unnecessary way.

A simpler and more direct technique may be employed by approaching the root apex from the palatal aspect. The advantages are threefold: First, the approach is the most direct, providing easy access and visibility. Second, the general contour of the ridge is preserved to a large degree when the buccal plate and interseptal bone are left intact. Third, the lingual alveolar plate is thicker and stronger than the buccal plate and can therefore be used as a fulcrum in an elevator technique, which further simplifies the problem.

Before undertaking operative intervention on the palatal surface of the maxilla, one should be familiar with the anatomy of the area involved. The greater palatine foramen is situated in front of the hamular process at the level of the third molar, usually at the junction of the hard palate and alveolar

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process. It transmits the descending palatine vessels and the anterior palatine nerve. The artery emerges from the foramen as the greater palatine artery and runs anteriorly in a groove on the palate. The terminal branches run forward to the incisive canal, where they anastomose with the sphenopalatine artery.[1] The mucoperiosteum of the palate is fairly thick, containing large amounts of connective tissue. Although it is tightly fixed to the underlying bone, it is not difficult to handle, because of its thickness and because it is not easily torn.

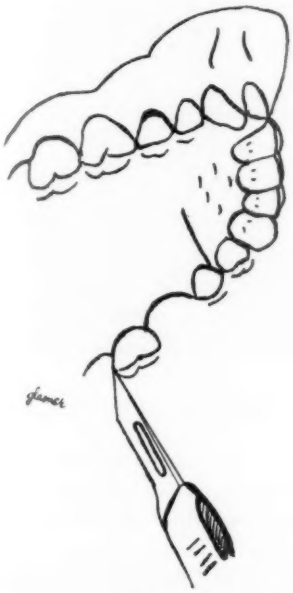


Fig. 1.

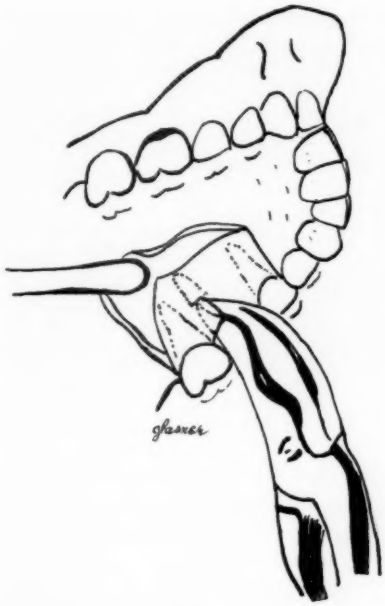


Fig. 2.

Fig. 1.—Incision employed for making flap.
Fig. 2.—Removal of lingual alveolar plate with rongeur exposing occlusal portion of apex.

In operating from the palatal aspect, due consideration should be given to the integrity of the major palatine artery. It is not necessary to incise the mucoperiosteum down to the level of distribution of the vessels and nerves in order to achieve adequate access. The incision on the palate can begin one tooth anterior to the involved tooth and terminate slightly below the occlusal level of the remaining root. This will place the terminal part of the incision above the greater palatine vessels (Fig. 1). After retraction of the flap, the lingual alveolar plate is nipped off in one piece down to the occlusal level of the root with a thin-beaked rongeur (Fig. 2). With the occlusal level of the root visible, enough bone may then be removed with a bone burr on the mesial aspect of the root to expose the mesial surface of the apex for a short distance. The opening in the bone should be wide enough to accommodate a small straight

elevator and deep enough in an apical direction to expose sound root structure capable of withstanding stress. The elevator is then inserted in the opening thus created, and the root surface is engaged (Fig. 3). The lever is then twisted

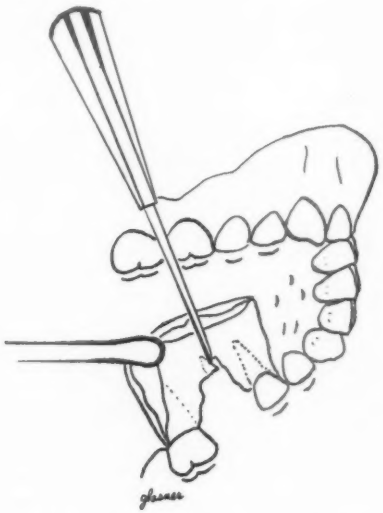
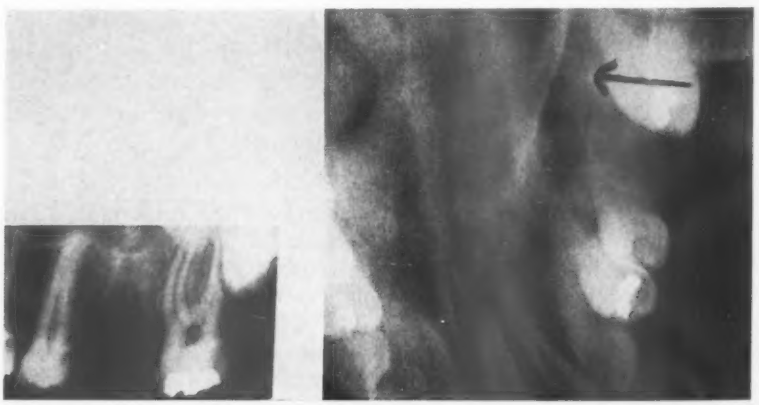


Fig. 3.—Bone removed on mesial aspect of apex, permitting use of elevator.

in an occlusal direction, with the mesial surface of the palatal alveolus serving as a fulcrum. Additional breakage of the root apex is not likely to occur if sufficient sound tooth structure has been exposed and an adequate fulcrum is

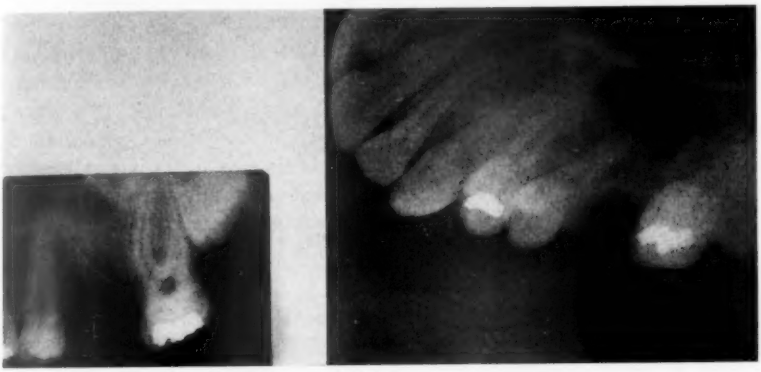


A. *B.*

Fig. 4.—*A*, Intraoral view showing fractured lingual apex high in alveolus. *B*, Bird's-eye view showing fractured apex.

present. After filing and débridement of the bony process, the mucoperiosteum is returned to its original position. One suture may be employed to hold the flap in place.

Roentgenograms illustrating the appearance of a lingual apex fractured and remaining high up in the alveolus, posing a fairly common and sometimes vexing problem in removal, are shown in Fig. 4, *A* and *B*. Fig. 5, *A* and *B*, shows postoperative views taken following the removal of the apex by the technique outlined.



A. *B.*

Fig. 5.—*A*, Intraoral view showing apex removed. *B*, Bird's-eye view showing apex removed.

SUMMARY

A method for removal of the lingual apex of an upper molar has been presented. A flap type of operation is employed to remove the root from a palatal approach which provides good access and visibility.

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515 Ocean Ave.

AUTO-EXTRACTION

David N. Plessett, D.D.S., York, Pa.

A 9-YEAR-OLD white girl was brought to this office by her parents. Clinical examination revealed that she was of normal body weight and size for her age but was suffering from an active measles outbreak. The face and body were covered with a maculopapular cutaneous rash, but no Koplik spots were visible intraorally and the disease apparently was in its later stages. The vermilion borders of the lips were bruised and slightly lacerated, and while the mucous membranes, palate, and gingivae were of normal color and architecture, the tongue was contused at the apex on the lingual side with tiny areas of ecchymosis.

The mandibular permanent central incisors were missing, with sockets containing freshly formed blood clots (Fig. 1). Only the lateral incisors and sixth-year molars were present in the mandible, while all incisors, some decayed deciduous molars, and the sixth-year molars were present in the maxilla.

Although no abnormal behavior had been noted by her parents, prior to the incident which brought her to this office, the girl had a history of having worked her maxillary deciduous cuspids from her mouth. Her I.Q. was within normal limits. On entering the child's room that morning, the parents saw the girl sitting in bed, her bedclothes stained with blood, and her two lower central incisors in her hand. Interrogation of the youngster failed to elicit an explanation of what had happened, but she implied that she had worked the teeth from her mouth by wiggling them. Further questioning failed to reveal a reason for this action. The parents were referred to a mental health clinic for investigation of this abnormal act.

It was decided to attempt a replantation of the extracted incisors. Under aseptic conditions, the root canals were enlarged, sterilized, and filled with gutta-percha points and silver cement. Sterilization of the teeth was effected with benzylkonium chloride, the teeth were smoothed, and the apices were shortened. Under local anesthesia, the clots were removed with a curette and the teeth were placed in the sockets and splinted to the adjacent lateral incisors by means of stainless steel ligature wire reinforced with a heavier-gauge orthodontic wire. A roentgenogram taken immediately following the operation showed good adaptation (Fig. 2). The teeth were disoccluded.

After three weeks a thorough curettage of the affected areas was performed and this was followed by a six-week interval, at the end of which a further

roentgenographic study with gutta-percha points showed no apparent attachment and beginning apical radiolucencies (Fig. 3). Suppuration was visible clinically. Resorption of the roots had become apparent, and some mobility of the entire splint was evident. Curettage was performed again, and after another six-week interval the gingival tissues had become congested (Fig. 4).

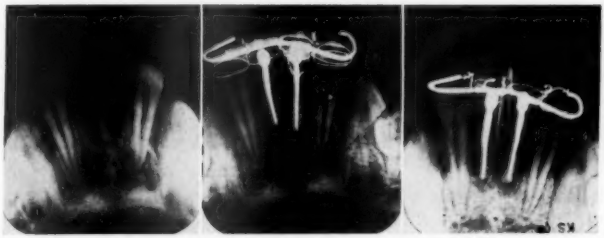

Fig. 1. Fig. 2. Fig. 3.

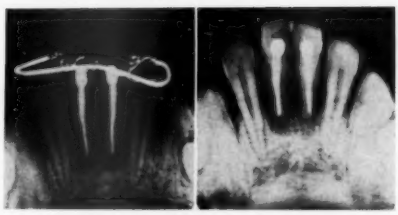

Fig. 4. Fig. 5.

Root resorption had progressed, and further mobility was demonstrated (Fig. 5). Inability to distribute the stress over enough teeth prompted removal of the replanted central incisors in order that the lateral incisors might maintain their integrity.

1243 East Market St.

ORAL MEDICINE

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MUCORMYCOSIS OF THE PARANASAL SINUSES AND THE MAXILLA

Philip S. Faillo, Lieutenant Colonel, DC, USAR, Herbert P. Sube, Lieutenant Colonel, MC, USA,** and Ned H. Anderson, Lieutenant Colonel, DC, USA.****

WHAT is mucormycosis? According to Baker,[2] mucormycosis is a disease caused by certain fungi (especially *Rhizopus*, which is a common laboratory contaminant and not ordinarily pathogenic). The fungi enter the nose and produce sinusitis and orbital cellulitis, they penetrate arteries and produce massive thrombosis of arteries, and later they invade the veins and lymphatics. Does this happen in all persons? No. So far, this disease occurs in susceptible patients who have uncontrolled diabetes or leukemia or who have been overtreated with antibiotics, antileukemic drugs and cortisone.

Mucormycosis is said to be a new disease in the United States. A report of the cerebral form[5] appeared in 1943, and one of the pulmonary form[3] appeared in 1948. However, some cases have been observed in the United States, Canada, and England. It has been encountered in nearly all cases as a complication of the aforementioned diseases. Diabetics who formerly developed micrococcic (staphylococcic) infection may now contract this new fungus infection.

Thirteen fatal cases have been reported since 1943.[3] Patients have been of all ages from 5 months[4] to 65 years, and sex and race have been irrelevant factors. Ten of the thirteen presented orbital and paranasal involvements; carotid artery thrombosis was frequent, and in seven there was existing diabetes mellitus.

In the case to be reported here there was obvious indication that the invasion began with the paranasal sinuses, and later there was some extension to the orbit.

CASE REPORT

In the early part of September, 1957, Major R. C., a 50-year-old white man, a retired major in the United States Army, was admitted to the medical department of the United

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***Orthodontist, Dental Clinic, United States Army Hospital, Fort Benning, Georgia.

States Army Hospital at Fort Benning, Georgia, with a diagnosis of orbital and maxillary cellulitis. Cavernous sinus thrombosis was suspected. On admission, the patient presented severe proptosis of the right eye with paralysis of the third through the seventh cranial nerves. Examination of the mouth showed a midline line of demarcation suggesting necrosis of the right side of the hard palate. There was no evidence of retinal vein engorgement, and it was presumed at this time that the case was one of arterial rather than venous involvement. The patient was subsequently referred to the eye, ear, and nose department for treatment of a hopeless condition. The chief of service of the EENT department made a careful study and then asked the oral surgery service (dental department of the hospital) to help as consultants. It was decided that we (EENT and oral surgery departments) would do a Caldwell-Luc operation on the patient's right maxillary sinus for débridement and biopsy. It was also noted at this time (Oct. 4, 1957) that there was considerable necrosis and tissue destruction overlying the hard palate. At operation the maxillary sinus was found to be filled with a mass of foul, greenish, necrotic substance. Biopsy sections were taken at the border of viable tissue and in the region of the junction of the soft palate. Immediate frozen sections revealed much necrosis and a bizarre pattern of inflammatory cells, from which nothing could be concluded from histologic study. On Oct. 10, 1957, it was decided that a resection of the necrotic exposed right maxilla (Fig. 1) would be performed in the hope of establishing still further histologic evidence of pathology.

Fig. 1.—Preoperative photograph showing necrotic maxilla. *a*, Necrosis.

Our pathologist hinted a possible fungus invasion. Resection of the maxilla was rather simple because of the destruction of the large arteries, which appeared thrombosed and necrotic. A débridement was carried out very carefully until we reached an area in the region below the middle fossa of the brain. Because of the proximity to the internal carotid artery, further débridement was accomplished with the use of a proteolytic enzyme to digest residual necrotic bone which might act as a nidus. The proteolytic enzyme used was papain, or an extract from the papaya fruit. This substance was used in a form which was prepared by Faillo[6] in earlier experiments in 1948—powdered papain in combination with a vehicle known as Carbowax 1500.*

*In the *Merck Index*, sixth edition (published in 1952 by Merck & Co., Inc., Rahway, New Jersey), Carbowax compounds are described as unctuous waxlike solids which dissolve in water, forming transparent solutions. Carbowax belongs to the series of solid polyethylene glycols. It will not deteriorate on storage and will not support mold growth.

Fig. 2.—Healed defect. *a*, Epithelial growth.

Fig. 3.—Microscopic demonstration of fungus. *a*, Fungus.

It was so compounded because papain was speedily inactivated when used by itself. Papain particles enveloped in a coating of water-soluble Carbowax 500 can be controlled to act slowly and effectively when applied on gauze to the area treated.

Packs containing the proteolytic enzyme and daily irrigations with a saline suspension of mycostatin resulted in a clean and healthy wound covered with granulation. This procedure was continued for approximately two months (Fig. 2).

To ensure proper nutrition, a Levin tube was first inserted. It did not give enough nutrition to control the diabetes, however, and a loss of weight was noted. The lost portion of the maxilla was built up with additional packs of petrolatum gauze which provided enough bulk that the patient could wear his denture, which provided a means of mastication and helped in swallowing.

All antibiotics were immediately stopped when the fungus was discovered following resection and biopsy with histologic study (Fig. 3).

Fig. 4.—Prosthesis.

The only medication that the patient received was potassium iodide, and his diabetes was brought under control with daily administration of insulin. Within three months, the patient regained 24 pounds of body weight. It was thought that perhaps a split-thickness skin graft would have to be used as a lining to cover the defect if no epithelization occurred; however, enough islands of epithelium had remained among the granulation tissue that within a short time full epithelization of the entire defect took place (Fig. 2). The remaining problem was to construct a prosthesis attached to the patient's full upper denture to close off the large defect in much the same way one would construct an obturator for cleft palate. Our orthodontist was engaged in this task, and a successful prosthesis was constructed (Fig. 4) which materially aided the patient's speech and deglutition of a normal

diet. We noted with pleasure the rise in the patient's morale and the genuine interest which he took in the success of his recovery to a normal rehabilitation (Fig. 5). The problem of self-care and self-cleaning of the defect by the patient required some training, since the normal crusting which is found in the nose was also accumulating in the defect. To this end, a 1,000 c.c. douche can and tip were daily filled with warm saline and the patient was instructed to cleanse the defect by seating himself on a stool and running the solution from the nasal opening into the defect and then out from the mouth. After two weeks' training, he was quite competent to maintain his own oral hygiene.

Fig. 5.—Front view of patient showing healing and improved vascularization of skin lesion.

The *Rhizopus*, rather than the *Mucor*, appears to be the usual cause of human mucormycosis. *Rhizopus* grows rapidly, and the rhizoids appear in microscopic structure.[5] The predominating feature of mucormycosis is the presence of broad, branching, predominantly nonseptate hyphae[1] (Fig. 3).

The diagnosis is sometimes difficult and is usually made by examination of tissue specimens taken at biopsy. Since *Rhizopus* is a common laboratory contaminant, the recovery of this organism in culture is not deemed to be diagnostic. The disease is treated by removal of the nidus followed by iodine therapy[1] and the cessation of all antibiotics. Locally, the proteolytic enzyme therapy described will suffice to complete débridement of critical areas during

healing; myeostatin and saline solution suffice for irrigations. Again, it is imperative that all antibiotics, cortisone and corticotropin therapy should be discontinued or at least re-evaluated. If the patient is a diabetic, immediate control should be attained. In this case, the patient is at this date (March 4, 1958) in such good control that no insulin has been needed in the past month. Weight gain has come about in a normal and healthy manner, and a good rehabilitation has been the reward. With the dental prosthesis, he has a full normal masticatory function and a good deal of nerve regeneration, especially of the fifth and seventh cranial nerves, has also been obtained. The skin of the face on the affected side has improved with regeneration of collateral blood supply (Fig. 5). When eyeball function improves (the excursion of the eyeball toward the outer canthus of the eye) the eyelid, which had been previously closed by a tarsorrhaphy in order to protect the eye, will be reopened.

In conclusion, the diagnosis of this disease must rest upon demonstration of the fungus in the lesions, and treatment is dependent upon the careful consideration of all of the underlying predisposing factors.

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CHRONIC FAMILIAL NEUTROPENIA, WITH MARKED PERIODONTAL LESIONS

Report of a Case

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THE normal range of total leukocytes is 4,000 to 11,000 cells per cubic millimeter of blood, and the normal range of neutrophil cells is 1,500 to 7,500 per cubic millimeter. Counts falling below the lower limits of normal constitute leukopenia and neutropenia.[1] These conditions commonly occur together as acute, chronic, or recurrent. Chronic leukopenia has a variety of well-known causes.[1] The case to be described in this article was associated with none of these, however, and appeared to be idiopathic. There seems to be no such case described in the dental literature. In the medical literature Robertson and associates[1] described a case of chronic idiopathic neutropenia, but the patient showed no increased susceptibility to infection as the one to be described here did. Reimann and de Berardinis[2] described a series of cases of chronic neutropenia with oral lesions, but in each case the neutropenia was cyclic.

CASE REPORT

A boy, aged 14 years, 7 months, reported on March 23, 1955, because of an unusual mouth condition which had troubled him for some years and appeared to be getting worse.

Clinical Examination of the Oral Cavity.—A marked hyperplastic gingivitis of a bright red jellylike appearance was found throughout the mouth, both lingually and labially (Fig. 1). In some areas the crowns of the teeth were half-covered by the masses of interproximal

Fig. 1.—Highly inflamed jellylike hyperplastic gingival tissue. Note subgingival calculus and materia alba (lower incisors). March, 1955.

gingival tissue which bled readily when touched. There were much debris and calculus and marked fetor oris, although there had obviously been an attempt to keep the teeth clean. There was pocket formation generally throughout both jaws, with bone loss quite marked in some areas. This is quite evident in the roentgenograms (Fig. 2). The lower anterior teeth showed some mobility.

On March 31, 1955, hematologic findings were as follows:

Red blood cells	3,990,000
Hemoglobin	11.5 Gm./100 ml. blood
Color index	0.97
Anisocytosis	+
White blood cells	4,100
Neutrophils	4% 164/c.mm.
Eosinophils	12% 492/c.mm.
Lymphocytes	56% 2,296/c.mm.
Monocytes	25% 1,025/c.mm.
Basophils	1% 41/c.mm.
Myelocytes	2% 82/c.mm.

This shows a marked neutropenia and monocytosis.

Urinalysis on March 24, 1955, revealed the following:

Reaction	Neutral	Specific gravity	1016
Albumin	-ve	Pus	-ve
Blood	-ve	Bile	-ve
Sugar	-ve	Acetone	-ve

Medical Examination (April, 1955).—The patient was 5 feet, 1 inch tall, and he weighed 90 pounds. He was a healthy, good-looking boy. There was some finger clubbing but no enlarged glands. His chest and heart were normal, and his blood pressure was 100/70 mm. His spleen was just palpable on full inspiration. His mouth was as described, and his fauces appeared normal. Urine showed a specific gravity of 1014, with no sugar and no protein.

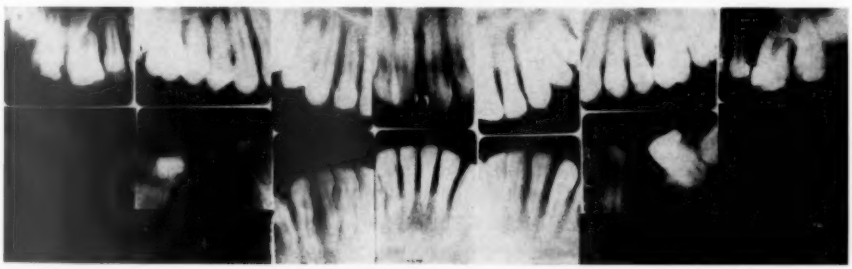

Fig. 2.—General bone loss, especially marked in upper posterior region. There are also carious lesions. March, 1955.

Medical History.—The patient was born in 1940, two weeks prematurely, and his mother had rubella in the seventh month of pregnancy. At 10 months he developed ulcers in the mouth; these recurred at two- to three-week intervals until quite recently, and they were particularly severe during tooth eruption. With the eruption of the deciduous molars, an abscess developed in the right cervical lymph glands. During 1942, at the age of 2 years, 4 months, the patient had a gross infection of the lips and was admitted to a hospital when his neutropenia was found. During 1943 he was treated on numerous occasions for sore throat, infected fingers, ulcers of the tongue, and many other infections. In 1952, while hospitalized for a tonsillectomy, he developed a respiratory illness with unusual shadow which

was subsequently considered to be an unresolved pneumonia. In 1953 he had an attack of pyelonephritis, and later he suffered a polyarthritis (of doubtful diagnosis) which lasted about two weeks. In this year he had a tonsillectomy and was then much better.

Throughout his illness he has suffered persistent inflammation of the gums with hypertrophy, bleeding, and fetor oris. In the past year or so his general health has been very much improved, although he has been subject to recurrent respiratory attacks lasting a week or so. *Apart from this, his only disability is the condition of his gingivae.* In addition to his recurrent infections, he has had measles, mumps, chickenpox, and scarlet fever without complications.

Hematologic investigations (from 1946): There has been a striking neutropenia at various times throughout this period, the neutrophil count being as low as 46 per c.mm. and the leukocyte count as low as 2,500 per c.mm. At other times the leukocyte count has been as high at 14,000 per c.mm. There has been no apparent correlation between the neutropenia and any other factors. The hemoglobin has varied from 10 to 12 Gm. per cent.

Family History.—There is a strong family history of neutropenia. The patient's father (now edentulous), one sister, and one brother suffer from the condition, though not as severely as he does. A sister, aged 5 months, has so far shown no sign of the disease. There is no history of the condition in the grandparents, nor in the twenty-one cousins of his own generation.

Fig. 3.—Gingivectomy has been performed on upper right quadrant. Patient's oral hygiene has improved. There is no materia alba on labial aspect of lower incisors. Hyperplasia appears more marked, however. June, 1955.

Treatment.—In spite of his medical history, it was decided that the patient should be given periodontal treatment to see if some improvement could be made in his mouth condition. A prophylaxis was attempted on April 4, 1955, which, under the circumstances, was by no means complete, and the patient was instructed in a method of toothbrushing which would help to free the mouth of debris and improve his oral hygiene. When he returned on April 12, 1955, there was less odor from the mouth and the teeth were much cleaner. It was then decided to attempt gingivectomy. This was performed on the upper right quadrant on May 16, 1955, and was accompanied by hemorrhage which was stopped with pressure. There was found to be considerable bone loss in the $\underline{7\ 6}|$ area. Healing was slow, and on June 10, 1955, the gingivectomy area looked very good (Fig. 3). The lower anterior teeth seemed to be more mobile and more separated, however, and they appeared to have more tissue overgrowth. Roentgenograms of the lower anterior teeth (Fig. 4) had been taken on May 30, 1955. Admittedly, the angulation here is slightly different from that shown in Fig. 2, but there does appear to be more separation of the teeth and a clearer view is seen of the bone

loss. (During this month plasma protein estimations showed a considerable increase in the gamma globulin fraction, no doubt reflecting an increased production of antibodies to compensate for the lack of phagocytic activity of the blood.)

The patient did not keep his next appointment, as he was ill with another attack of pneumonia which was probably viral in origin and from which he soon recovered. Throughout this illness his neutropenia was evident.

The patient returned on July 12, 1955. The gingival condition around the lower teeth looked worse, and the tissue was regrowing interproximally in the upper right quadrant. The patient seemed psychologically unable to cope with the situation as far as home care was concerned. It was then decided that all his teeth should be extracted.

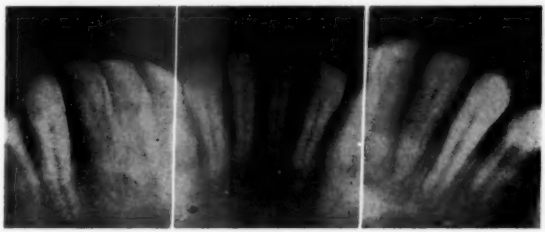

Fig. 4.—Spacing of teeth seems greater (see Fig. 2). May, 1955.

A hematologic report in August, 1955, revealed the following:

Hemoglobin	11.0 Gm. per cent
Leukocytes	3,900/c.mm.
Neutrophils	4 per cent 156/c.mm.

All teeth except the four third molars, which were not yet erupted, were extracted between August, 1955, and January, 1956. The patient was given xylocaine with a penicillin cover and vitamin K. There was some hemorrhage with slight fever after some extractions, but healing was otherwise uneventful. Full upper and lower dentures were inserted, and between April, 1957, and June, 1957, the four third molars were extracted.

DISCUSSION AND SUMMARY

A case of chronic familial neutropenia is presented. This case showed marked periodontal lesions involving both gingival tissue and supporting bone. The etiology of the neutropenia was unknown, and it was present in other members of the patient's family. Except for the oral lesions, the patient had no other persistent disease, although from time to time he suffered from pulmonary and upper respiratory tract infections and showed the usual increased susceptibility to infections. Occasionally, cortisone has been found to increase the neutrophil count, but this would have required prolonged therapy at considerable expense, with the results by no means certain. Therefore, the tooth extractions were not delayed, in view of the marked bone destruction and especially in view of the fact that periodontal treatment had been undertaken without success.

I wish to thank Dr. Ralph Reader for his help and the medical reports, Dr. E. R. Magnus for undertaking the extraction, and Mr. G. M. Withycombe for taking the Kodachromes from which the illustrations were made.

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PHARMACOLOGY AND THERAPEUTICS

ANTIBIOTIC LEVELS IN SALIVA OBTAINED WITH A NEW ANTIBIOTIC: THIOSTREPTON TROCHE STUDIES

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THIOSTREPTON is a new, extremely active antibiotic with a penicillin type of spectrum of activity. It has been introduced to the professions rather recently.[1-3] This antibiotic principal is obtained from an unidentified strain of *Streptomyces* isolated from a New Mexico desert soil sample. Its in vitro spectrum is essentially limited to the gram-positive cocci and the *Neisseriae*. Initial disc sensitivity studies indicate also that the spectrum is very similar to that of penicillin.[4, 5] The solvent requirements, isolation, and characterization of crystalline Thiostrepton, paper chromatography studies, stability, and minimal inhibitory concentration of Thiostrepton against various microorganisms have been described in detail.[1-3]

The antibiotic was observed to be primarily bacteriostatic. It was found to be stable in simulated gastric and intestinal juices, feces, urine, and blood plasma. It can be assayed by either the tube dilution method or the agar diffusion method.

Disc sensitivity studies[4, 5] and tube dilution sensitivity studies[6, 7] all suggest a useful spectrum of activity and adequate potency of this new antibiotic. Blood chemistry, urinalysis, and hematologic studies also suggest a lack of deleterious action of this antibiotic on body function.[8]

Resistance of the type and degree of that developed to penicillin by organisms belonging to once highly susceptible species (now encountered with increasing frequency) is not as yet in evidence with Thiostrepton, due most probably to the new agent's limited clinical use as of the present. Because of the marked insolubility of this compound and its lack of absorption through the gut, no parenteral or systemic oral dosage form is anticipated. It can thus be conjectured that systemic sensitization to the use of this antibiotic should also be

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Thiostrepton troches for this study were supplied by the Squibb Institute for Medical Research, New Brunswick, New Jersey.

minimal. It is also felt that administration of Thiostrepton would not contribute to the production of microorganisms resistant to an antibiotic which later might be needed systemically. On the basis of the above, Thiostrepton has been suggested to be of value (primarily) in the treatment or prevention of superficial infections following its topical application to various accessible sites, such as the mouth.

In the study to be described here, Thiostrepton has been incorporated into troches in a concentration sufficient to portend effective action on a topical basis, as opposed to the relatively minimal concentrations of other antibiotics previously available in topical antibiotic preparations. The probable relative absence of systemic sensitization due to the lack of gut absorption of the antibiotic will, it is hoped, permit effective antibacterial action at such relatively higher antibiotic concentrations as may be required to eradicate superficial intraoral infections.

The absence of a disagreeable taste has permitted the use of this antibiotic in formulations without the inclusion of those excipients which may have contributed to the evidences of side reactions often seen with other topical (oral) antibiotic preparations.

As noted above, this antibiotic is characteristically markedly insoluble. This property might be detrimental in the event that it created tissue irritation but on the other hand, it might be a useful attribute if it were well tolerated by the tissues. Insolubility might thus contribute to more prolonged and higher saliva levels.

This study was undertaken to investigate the levels of Thiostrepton which were established and maintained in human saliva during the course of the routine administration of this antibiotic in troche form.

METHODS

Twelve adult male volunteers (aged 22 to 45 years), all graduate students or members of the staff of the School of Dental and Oral Surgery, Columbia University, were studied as follows:

A control pretreatment sample (Sample A) of unstimulated saliva was obtained from each subject. Immediately thereafter, a single troche containing 10 mg. of Thiostrepton was administered for dissolution in the mouth. After fifteen minutes, a second sample of saliva (stimulated, owing to the presence of the troche within the mouth) was obtained (Sample B). Approximately a three-minute collection specimen of saliva was obtained in each instance throughout the study. The fifteen-minute initial time period of study was chosen inasmuch as it seemed to represent the time at which the peak salivary level of Thiostrepton probably might be anticipated since from that time on the physical quantity of troche present in the mouth markedly decreased in size and volume.*

*The time during which the troche used in this study remains in the mouth during the normal activity of the person could be considered to be roughly fifteen to twenty-five minutes. When an effort is made not to move the troche about the mouth unnecessarily, the period during which the troche can be expected to last, however, may be extended to one hour or even longer, whereas if the troche is moved about actively, chewed, etc., this period is markedly reduced.

Approximately one hour after the control saliva specimen was obtained (forty-five minutes after the salivary specimen was obtained at the anticipated theoretical peak of Thiostrepton concentration in the saliva and approximately thirty-five minutes after it was estimated that the troche had disappeared in mouths of the subjects), a third sample (Sample C) of unstimulated saliva was obtained. This specimen represents primarily the thirty-five-minute post-therapy level of residual Thiostrepton in the saliva. Immediately after the third specimen was obtained, a second troche was administered, and fifteen minutes later the final (fourth) sample of stimulated saliva (Sample D) was obtained.

Following collection, all saliva samples (totaling forty-eight) were coded and deep frozen until they could be assayed (still coded and blind) for Thiostrepton activity by an assay procedure reported previously.[1] All assays were performed by the Department of Microbiology of the Squibb Institute for Medical Research, New Brunswick, New Jersey.

RESULTS

All of the data obtained in this study appear in Table I.

Our findings suggest that salivary levels ranging from 15 meg. per milliliter to 690 meg. per milliliter and far in excess of the minimal inhibitory level[1] determined for susceptible organisms are obtained during the period of dissolution, and that even 35 minutes following dissolution of the troche significant levels of Thiostrepton, ranging from 1.8 meg. per milliliter to 98 meg. per

TABLE I. SALIVARY LEVELS OF THIOSTREPTON ASSAYED AT VARIOUS INTERVALS BEFORE, DURING, AND AFTER DISSOLUTION OF TWO 10 MG. THIOSTREPTON TROCHES IN THE MOUTH

PATIENT NO.	ZERO TIME, AFTER WHICH A THIOSTREPTON TROCHE WAS INSERTED INTO THE MOUTH SAMPLE A MCG./ML.	15 MINUTES AFTER INSERTION OF FIRST TROCHE SAMPLE B MCG./ML.	1 HOUR AFTER INSERTION OF FIRST TROCHE. 35 MINUTES, APPROXIMATELY, AFTER TOTAL DISSOLUTION OF THE FIRST TROCHE. AFTER THIS SAMPLE WAS TAKEN, A SECOND TROCHE WAS ADMINISTERED SAMPLE C MCG./ML.	15 MINUTES AFTER INSERTION OF SECOND TROCHE SAMPLE D MCG./ML.
1	Less than 0.5	200	1.8	40
2	Less than 0.5	470	98.0	30
3	Less than 0.5	150	28.0	85
4	Less than 0.5	210	9.0	160
5	Less than 0.5	420	9.0	31
6	Less than 0.5	690	13.0	96
7	Less than 0.5	15	Less than 15.0	54
8	Less than 0.5	170	3.6	89
9	Less than 0.5	400	12.0	260
10	Less than 0.5	290	6.0	170
11	Less than 0.5	47	Less than 2.0	28
12	Less than 0.5	28	Less than 2.0	20
Mean concentration of Thiostrepton:	Less than 0.5	257.5	15.0	88.6

milliliter remain (in nine of twelve subjects), levels which are substantially in excess of the minimum inhibitory levels required (see below) for susceptible microorganisms. A mean figure of 257.5 meg. per milliliter was obtained fifteen minutes after insertion of the first troche. A mean figure of 15.0 meg. per milliliter was obtained one hour after insertion of the first troche, approximately thirty-five minutes after dissolution of the first troche. A mean figure of 88.6 meg. per milliliter was obtained fifteen minutes after insertion of the second troche.

On the basis of these findings, it can also be concluded that Thiostrepton is adequately stable in human saliva.

DISCUSSION

The data reported here should be viewed against a background of the minimum inhibitory concentration of Thiostrepton required to demonstrate its bacteriostatic activity. These values have been reported, on the basis of in vitro studies, to be as shown in Table II.

TABLE II

ORGANISMS	MCG./ML.
Micrococcus pyogenes var. *aureus* 209P	0.024
Micrococcus pyogenes, Cahill strain (penicillin-resistant)	0.033
Micrococcus pyogenes (14 strains resistant to one or more of the following antibiotics: penicillin, streptomycin, chlortetracycline, oxytetracycline, chloramphenicol, and erythromycin)	0.047-0.125 (Average—0.07)
Streptococcus pyogenes C203	0.003
Bacillus subtilis	0.03
Streptococcus faecalis	0.06
Lactobacillus acidophilus	0.06
Clostridium septicum	0.03
Mycobacterium tuberculosis var. *bovis* BCG	3.0
Aerobacter aerogenes	50.0
Escherichia coli	50.0
Pseudomonas aeruginosa	50.0
Salmonella schottmuelleri	50.0
Shigella sonnei	50.0
Shigella dysenteriae	30.0
Proteus vulgaris	50.0
Neisseria gonorrhoeae	0.3
Diplococcus pneumoniae type 2	4.8
Klebsiella pneumoniae	50.0

Essentially, the above findings have been confirmed in our studies[6, 7] on various of the pathogenic organisms listed in Table I, which were obtained from clinical pathologic specimens.

No clear-cut explanation is offered to explain why the salivary levels of Thiostrepton in the fourth run (Sample D) were, as a group, lower than in the comparable second run (Sample B). It is believed that whatever unexplained difference is noted stemmed more likely from a different manipulation (including chewing and movement) of the troche in the mouth of the various persons or as the result of other actions, such as swallowing, reluctance to

swallow the medication at the time of the first administration of the troche, the later time of day with attendant thirst or hunger, etc. rather than from any difficulty in the assay procedure.

SUMMARY

In order to study the saliva levels of a new antibiotic, Thiostrepton, which might be obtained following the administration of troches containing 10 mg. of this antibacterial substance, salivary specimens were assayed before, during, and after the administration of Thiostrepton troches to twelve adult male volunteer subjects. Our findings suggest that, in terms of the minimum inhibitory concentration of Thiostrepton against various susceptible microorganisms, saliva levels far in excess of this minimum inhibitory level are obtained during the period in which the troche is not yet completely dissolved and that the saliva Thiostrepton levels some thirty-five minutes after the troche had dissolved were also well in excess of the minimum inhibitory concentration in at least nine of the twelve subjects.

The authors express their gratitude to Miss Evelyn Wirth, Mrs. Eleanor Weischadle, Mrs. Eileen Geller, and Mrs. Helene W. Kutscher for their assistance.

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A CLINICAL EVALUATION OF AN OXYGENATING AGENT

Preliminary Report

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INTRODUCTION

DURING the past several months, a new proprietary oxygenating agent* has been distributed to many of the dentists along with certain claims regarding its efficacy as an aid in the management of pyorrhea, necrotizing (ulcerative) gingivitis, stomatitis, gingivitis, etc. This preliminary study was undertaken to determine the validity of these claims and to evaluate the product on its own merits.

HISTORY

Chemical oxidizing and oxygenating agents have been used in dentistry for many years. In recent times, attention seems centered on those agents which liberate nascent oxygen upon coming in contact with the tissues. Hydrogen peroxide and sodium perborate are perhaps the best known and most widely used. There seems little doubt that properly used, oxygen-liberating solutions will (1) aid in the control of acute necrotizing gingivitis[1, 2]; (2) inhibit the growth of anaerobic bacteria[3]; (3) assist in the débridement of gingival tissues that have been curetted and sealed, thereby promoting more rapid healing.[4]

These observations refer to the topical effect of the oxygenating agents. It would seem that, according to our present knowledge, there is also a deeper effect caused by the introduction of oxygen into the tissue substance per se. Orban,[5] Kaiser,[6] Box,[7] and others have demonstrated the presence of oxygen within the tissue following the topical application of hydrogen peroxide. The effect of this intramucosal oxygen has not been determined. However, it is known that in the presence of degeneration the QO_2, or oxygen utilization of a given tissue, is reduced.[8, 9] This may be due to either an alteration in the biochemical oxidation enzyme system or a lack of available oxygen to the part. If it is due to the latter, then the addition of oxygen within the tissue substance would materially aid in the restoration of a condition of normalcy, at least temporarily.

From the School of Dentistry, University of California.

*Marketed under the name Amosan by the Knox Company, Los Angeles, California.

METHODS AND MATERIALS

The oxygenating agent studied was sodium peroxyborate monohydrate 70 per cent, buffered with anhydrous sodium bitartrate 30 per cent and flavored with menthol and oil of peppermint. It is packaged in individual units of 1.7 grams. Each patient was instructed to dissolve the contents of one package in approximately one tablespoonful of water and to use this as a mouth rinse for one and one-half minutes at a time. It was used three times a day, unless otherwise noted, each time after brushing the teeth.

Two separate series of patients were observed. In one the labeled oxygenating agent was used. In the second the unlabeled "double-blind" technique was followed.

In the first study the patients were issued a supply of the labeled proprietary preparation. The subjects were a random sample of patients presenting for periodontal treatment. They were of both sexes, ranging in age from 16 to 68 years. The clinical diagnoses of these patients included some twenty-three cases of periodontitis, three cases of acute necrotizing gingivitis, nine cases of gingivitis, five cases involving postsurgical healing and hemorrhage control, and two cases of chronic necrotizing gingivitis. In all cases, except the treatment of gingivitis, the rinse was used as an adjuvant to other dental manipulations. It was not intended to supplant or replace the usual curettage, scaling, or other surgical periodontal procedure.

In the second series forty-eight patients with periodontitis and gingivitis were studied. They were issued packages of powder identifiable only by a number, either "10" or "30." Neither the patient nor the investigator knew the contents. At the conclusion of this study it was found that Code 30 was the flavored sodium peroxyborate monohydrate buffered with anhydrous sodium bitartrate and that Code 10 contained sodium bicarbonate in place of the peroxyborate. Otherwise, the contents were the same. The same investigator followed each case.

RESULTS

In this pilot study, the results were based on the subjective reactions of the patient and the clinical judgment of the investigator. These were combined to give an over-all impression of "excellent, good, fair, or none." From the standpoint of effectiveness, "excellent" and "good" constituted positive results, while the others would be negative. Except where otherwise noted, the results were evaluated after one week.

Use of the Labeled Product.—The data are summarized in Table I. From it may be seen that *positive* findings are seen in the following:

1. Nineteen out of twenty-three cases of periodontitis.
2. Four out of five cases of control of postsurgical healing and hemorrhage control.
3. Three out of three cases of acute necrotizing gingivitis in one to four days.

TABLE I. USE OF LABELED SODIUM PEROXYBORATE THREE TIMES A DAY FOR ONE AND ONE-HALF MINUTES EACH TIME

CONDITION	NO. OF PATIENTS	RESULTS*		SIDE REACTIONS
		POSITIVE	NEGATIVE	
Periodontitis	23	19	4	2
Postsurgical hemorrhage healing	5	4	1	0
Acute necrotizing gingivitis	3	3	0	0
Gingivitis	9	4	5	1
Chronic necrotizing gingivitis	2	0	2	0
Total	42	30	12	3
Per cent	100	71	29	7

*Results were tabulated after seven days except for the cases of acute necrotizing gingivitis, in which results were noted in one to four days.

4. Four out of nine cases of gingivitis.

5. Neither of the two cases of chronic necrotizing gingivitis.

6. Total results showed thirty out of forty-two (or 71 per cent) positive.

Side reactions: In none of the subjects was an allergic reaction seen. However, three of the patients did manifest a sufficient burning sensation with tissue congestion to warrant discontinuing use of the mouth rinse. These untoward reactions were of a temporary nature and did not persist beyond the time of discontinuance.

TABLE II. DOUBLE-BLIND STUDY RESULTS AFTER ONE WEEK

CODE NO.*	PATIENTS	RESULTS		SIDE REACTIONS
		POSITIVE	NEGATIVE	
10	20	10 (50%)	10	0
30	28	22 (79%)	6	3 (11%)

*Code 10 is flavored sodium bicarbonate. Code 30 is flavored buffered sodium peroxyborate.

"Double-Blind" Study (Table II).—Among the subjects given Code 10 (flavored, buffered sodium bicarbonate), there were positive results in ten out of twenty cases (50 per cent). There was no evidence of irritation or other side reactions. There were positive findings in twenty-two of twenty-eight subjects given Code 30 (flavored, buffered sodium peroxyborate monohydrate) (79 per cent). Code 30 produced side reactions consisting of one case of severe irritation with small ulcers on the marginal gingivae and two cases of mild burning.

There was no evidence of bacterial or fungal overgrowth in either study, even after prolonged use of the product.

Consolidated Results (Table III).—Positive results were obtained in fifty-two of seventy patients (74 per cent). Untoward side reactions of a temporary nature were found in six of the seventy patients (8 per cent).

TABLE III. CONSOLIDATED RESULTS OF ALL PATIENTS USING THE BUFFERED SODIUM PEROXYBORATE MOUTH RINSE FOR ONE WEEK

	PATIENTS	RESULTS		SIDE REACTIONS
		POSITIVE	NEGATIVE	
Number	70	52	18	6
Per cent	100	74	26	8

DISCUSSION

The inherent weakness of a strict clinical evaluation is recognized. The double-blind test was done to attempt to eliminate one of the more obvious drawbacks, that of prejudicial observation.

The use of buffered sodium peroxyborate monohydrate in conjunction with the treatment of periodontal disturbances has been found to be moderately successful. While positive results were obtained in 74 per cent of the cases, it should be borne in mind that the double-blind study gave only 50 per cent positive findings for a bland mouth rinse. This is in keeping with findings made by Behrman and associates,[10] who found upward of 55 per cent improvement with either sodium chloride or sodium borate.

While side reactions were noted in 8 per cent of the patients, the character of the reactions was generally mild. In no case was any true allergy found, and the one severe reaction cleared within three days.

SUMMARY AND CONCLUSIONS

1. The effectiveness of a flavored, buffered sodium peroxyborate monohydrate mouth rinse was evaluated.

2. Positive findings were obtained in fifty-two out of seventy patients (74 per cent).

3. Results were uniformly good for acute necrotizing gingivitis.

4. A parallel study utilizing the double-blind technique indicated that 50 per cent positive results could be expected with a bland mouth rinse.

5. Six cases of untoward reactions were reported, representing 8 per cent of the patients. Of these, five patients experienced a slight to moderate burning sensation without any observable pathology. One patient reported irritation, and small ulcers were observed on the marginal gingivae. All six patients reported prompt disappearance of the symptoms upon discontinuance of the rinse.

6. No cases of allergy or bacterial or fungal overgrowth were observed.

7. On the basis of this preliminary study, the product has moderate effectiveness as an adjuvant to periodontal therapy.

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ORAL PATHOLOGY

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A CLINICAL REPORT ON SIX CASES OF AMELOGENESIS IMPERFECTA

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GENERALIZED defective enamel formation (amelogenesis imperfecta) is a relatively uncommon condition and occurs much less frequently than disturbances in enamel formation affecting only part of the dentition. Nevertheless, in recent years cases have been reported in the literature by Weinmann, Svoboda, and Woods,[7] Gustavson, Nystrom, and Stelling,[2] Rushton,[3] Tebo,[6] Stones,[5] Shear,[4] Weyman,[8] and Cameron and Bradford,[1] and there appears to be quite a wide range of clinical appearances covered by the term *amelogenesis imperfecta.*

Weinmann, Svoboda, and Woods[7] divide the condition into two types—enamel hypoplasia and enamel hypocalcification. The difference is thought to be due to the stage of enamel development at which the defect occurs. In enamel hypoplasia the enamel matrix appears to be imperfectly formed and, although calcification subsequently occurs in the matrix so that the enamel is hard, it is deficient in amount and very irregular in deposition. In enamel hypocalcification matrix formation appears to be normal so that the enamel is normal in thickness, but calcification is deficient and the enamel is soft. This soft enamel wears or chips away rapidly after eruption or, if not, stains readily from the mouth due to its increased permeability.[2] Consequently, the teeth are frequently yellow or brown in color when the patient is first seen, although they are not necessarily so when newly erupted. In many cases of hypocalcification the defect is more marked on the occlusal and incisal areas of the tooth crowns.

The enamel appears to be the only structure affected, and in general there are no other abnormalities.

Six cases of amelogenesis imperfecta, all occurring in children, are described here. Five different families are involved, and in two of them the condition is obviously hereditary. Both hypoplasia and hypocalcification are represented, and some of the features are unusual.

From Mount Vernon Centre for Plastic Surgery, Department of Jaw Surgery.

Case 1. Hereditary Enamel Hypoplasia.—An 11-year-old girl presented with an unusual form of hypoplasia, together with delayed and arrested eruption. She has an interesting family history, and the reported incidence of the hypoplasia in the family is shown in Fig. 1. The defect is obviously inherited via the father, but it appears to have missed his generation completely. Three of his five children, however, have hypoplastic teeth, and four of his brother's five children are similarly affected, while additional evidence supporting the inheritance is provided by the father's two marriages.

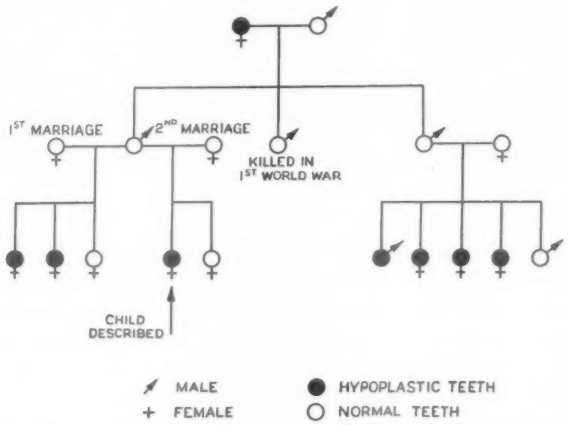


Fig. 1.—Case 1. Reported distribution of hypoplasia in patient's family.

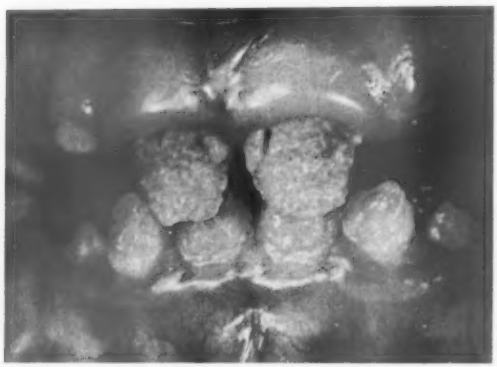
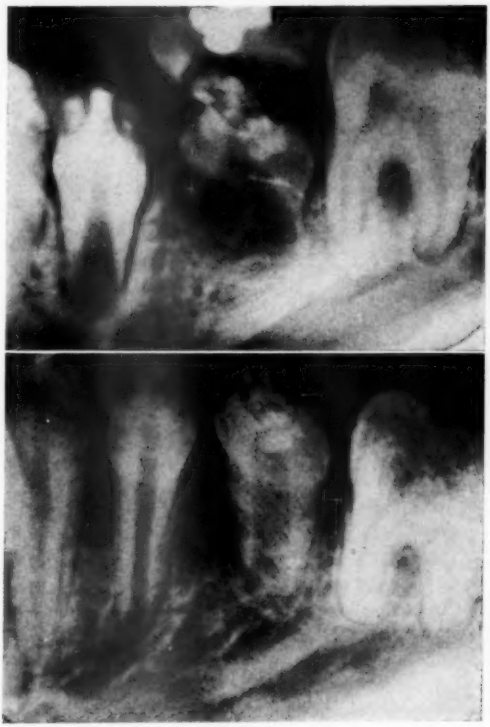


Fig. 2.—Case 1. Pitting type of hypoplasia.

This patient is the eldest child of the second marriage. She has a mixed dentition, and both deciduous and permanent teeth are hypoplastic. The hypoplasia is minimal on the deciduous teeth; the enamel on the occlusal surfaces of their crowns is missing, and the remainder of the enamel is thin, yellow in color, and rough in texture. On the other hand, the permanent dentition is markedly hypoplastic (Fig. 2). All the erupted teeth show confluent pitting of the entire enamel surface. The enamel is hard but very thin and is absent in the depths of some of the pits. The teeth are conical in shape, tapering incisally and occlusally, and are white in color except where the yellow dentine is exposed.

Fig. 3.—Case 1. Roentgenogram illustrating mottling due to hypoplasia. Note normally developing roots.

A.



B.

Fig. 4.—*A*, Case 1. Roentgenogram of unerupted $\overline{|6}$ at age 9; $\overline{|45}$ as yet unerupted. *B*, Two and one-half years later; $\overline{|6}$ still unerupted, showing resorption of crown and eruption of $|45$.

At present no staining has occurred and there are no carious lesions. Radiographically, the teeth have a mottled appearance corresponding to the varying thicknesses of the enamel (Fig. 3).

The child has been observed for over two years, and the state of the dentition at the age of 11 is as follows:

$$\frac{1 \mid 1}{21 \mid 12} \text{ erupted} \qquad \frac{5432 \mid 234}{543 \mid 345} \text{ erupting}$$

The first permanent molars have shown no signs of eruption at all during this period and are covered by large gum pads which occlude when the child closes her mouth. The roentgenograms (Fig. 4) show that the crowns of these teeth are apparently undergoing resorption, although the roots are developing normally and the stage of development is consistent with the patient's age. The eruption of other permanent teeth has been slow, although some progress has been made during the time this girl has been under observation.

The child does not show any other abnormalities, although she is rather small for her age.

Case 2. Hypoplasia of Unknown Etiology.—A girl, aged 11 years, presented with a hypoplasia affecting all the permanent teeth, but to a varying degree. No trace of a similar condition, either past or present, could be discovered in the family. There is no delay in eruption, and permanent teeth as far back as the second molar are erupted. When first seen, one deciduous tooth remained. This was normal in appearance, and as far as could be ascertained the deciduous dentition was not hypoplastic. This tooth has since been shed. The permanent teeth, on the other hand, are all hypoplastic, but the degree of hypoplasia varies

Fig. 5.—Case 2. Left mandible showing hypoplasia and enamel nodules.

from tooth to tooth. Thus, some of the incisors merely show white hypoplastic patches and are of normal shape, although the enamel is thin and frail looking, while other incisors are also notched incisally. Apart from $\overline{5}|$, which resembles the incisors, the premolars are the teeth most affected (Fig. 5). They have completely lost their normal morphology, and the enamel is missing over large areas on their crowns. Cervically, there is a rim of thin, almost normal enamel, and elsewhere nodules of enamel are present. There is a sharp demarcation between areas of enamel and areas in which the enamel is missing. Where exposed, the dentine is hard and yellow but with a somewhat roughened surface. The hypoplasia on other teeth varies between these two extremes, and the whole combines to give the mouth a "broken-down" appearance, although oral hygiene is good and caries is minimal.

In view of the unusual form of the hypoplasia and the absence of any report of the condition in the family, it was decided to make further investigations into this case. Wassermann and Kahn reactions were negative. Serum calcium, phosphorus, and alkaline phosphatase were within normal limits. When she was 12 to 18 months old, the child suffered from attacks of constipation severe enough to warrant enemas for their relief. She had measles, mumps, and chickenpox between 5 and 6 years of age and an attack of dysentery at 7 years of age.

Fig. 6.—Case 3. Hypocalcification affecting incisal end of crowns more than gingival end.

Fig. 7.—Case 3. Roentgenogram corresponding to photograph shown in Fig. 6. Note enamel indistinguishable from dentine but normally developing roots.

Cases 3 and 4. Hereditary Enamel Hypocalcification.—Two cases of hereditary enamel hypocalcification fairly typical of the condition are seen in a brother and sister. The defect has been transmitted by the father, who had similar teeth. Of the four children in the family, three show the defect. There is no delay in tooth eruption in these children.

The boy, aged 11, has no deciduous teeth remaining, and all the permanent teeth are affected (Fig. 6). On newly erupted teeth the enamel is yellow and soft, but where teeth

have been present in the mouth for some time the enamel is brown. In some places the enamel has been worn away, but elsewhere it is of normal thickness. In general, the incisal and occlusal parts of the teeth are the most affected and the lower incisors are not so severely affected as other teeth, but none of the enamel is clinically normal. Radiographically, it is difficult to differentiate enamel from dentine, but other tooth structures appear to be developing normally (Fig. 7). In this child oral hygiene and gingival condition are poor, much calculus is present, and there is a marked halitosis, but there are very few carious lesions.

The girl, aged 7 years, has a mixed dentition. All the deciduous teeth present are hypoplastic. Their enamel is reduced in thickness and is pitted and opaque. The permanent teeth are more severely affected and resemble the brother's. The enamel is missing in many places and, where present, it is rather soft. The distribution of the defect on the tooth crowns resembles the brother's, as do the roentgenograms, and again the oral hygiene and gingival condition are poor.

Fig. 8.—Case 5. Left mandible. Hypocalcification with brown-stained occlusal surfaces of crowns and demarcation line where enamel commences.

Case 5. Hypocalcification of Unknown Etiology.—A 12-year-old boy presented with a hypocalcification affecting all the permanent teeth. There is no report of a similar condition in his family, and there has been no delay in tooth eruption. All the deciduous teeth have been shed and, as far as is known, they were not affected. On the permanent teeth the defect is most noticeable on the incisal and occlusal third of the crowns; there is a somewhat sharper demarcation line than in the previous children and the enamel is harder cervically (Fig. 8). Roentgenographically, this is evident in that it is possible to distinguish enamel from dentine on the cervical margins of some of the teeth. On the affected areas much of the soft enamel has been worn away, and the cuspal and incisal areas of the teeth are rounded and stained a yellow-brown color. The majority of the remaining enamel is whitish and opaque.

When first seen, the second permanent molars were newly erupted and had a complete covering of enamel. This was soft and chalky over the occlusal surface and cusps and could be scraped away with an excavator. Over a period of two and one-half years' observation, however, much of this soft enamel has been gradually worn away and the teeth have become increasingly stained, so that they now resemble the other teeth in the mouth. In this child oral hygiene and gingival condition are poor, and gross calculus is present. There are two or three carious lesions but not as many as in a normal child of this age.

In the absence of a family history of the condition, this boy was also investigated for possible causes of the hypoplasia but with negative results.

Case 6. Hypocalcification of Unknown Etiology.—A case of hypocalcification affecting both deciduous and permanent teeth was seen in a 7-year-old girl. There is no report of a similar hypocalcification in the family, although the father has a localized hypoplasia affecting his incisor teeth. The deciduous teeth have no occlusal or incisal enamel; the rest of their enamel is rather rough in texture and stained yellow in places, but it is reasonably hard. The permanent teeth, on the other hand, are more severely affected with areas of hypocalcified enamel principally on the occlusal and incisal surfaces of the crowns but not entirely confined to these areas. The defective enamel is white, rough-surfaced, and opaque with light brown stained patches. The greater part of the labial enamel is missing on $\underline{1}|$, as is the majority of the occlusal enamel on $|\overline{6}$. The other teeth still have a complete enamel covering, although their incisal edges and cuspal tips are worn and rounded. The child has a high standard of oral hygiene, and no calculus is present. The radiographs present the usual picture of poorly differentiated enamel, but other structures appear unaffected. The lower right second premolar is congenitally missing.

DISCUSSION

The cases described illustrate the two broad divisions of amelogenesis imperfecta, although all of them show individual variations within the general description. As can be seen, both hypoplasia and hypocalcification may occur either with or without a history of the condition in the family. In cases with a family history, the condition appears to be inherited as a nonsex-linked Mendelian dominant, and usually one of the parents has similar teeth (for example, Cases 3 and 4). On the other hand, Case 1 is interesting, since there seems no doubt that the inheritance is through the father and yet the condition did not appear in his generation. Nevertheless, the majority of his children are affected, so this would not appear to be a recessive characteristic and it is unfortunate that the history cannot be traced farther back than the paternal grandmother to confirm this.

A comparison of the deciduous and permanent dentitions in these children shows that the incidence of amelogenesis imperfecta in the two dentitions varies and, while always present in the permanent dentition, is not necessarily found in the deciduous dentition. Where the deciduous dentition is affected, the defect is usually less marked than in the permanent dentition.

It is noteworthy that these six children show a remarkably low rate of caries incidence in spite of the deficient or poorly calcified enamel, the areas of unprotected dentine, and the rough irregular surfaces of the teeth which produce many stagnation areas. Otherwise, these children appear to be normal; no other ectodermal structures are affected, and in the two cases tested there were no abnormalities in the blood chemistry to account for the enamel defects.

Cases 1 and 2 (Figs. 2 and 5) differ quite markedly in clinical appearance and yet both are examples of hypoplasia. The pitting type of hypoplasia, as seen in Case 1, is uncommon. Weyman[8] described a child who had pitting over the whole of the enamel surface, but the teeth were of normal contour whereas in the case described above the teeth taper incisally and are not normal in shape. The hypoplasia shown in Case 2 is also unusual, both in appearance and in the fact that the enamel defects vary in severity from tooth

to tooth. These defects cannot be related to the time of formation of the teeth, however; otherwise, there would not be such a marked difference between the lower right second premolar and other premolars, since these teeth must have developed concurrently. It would appear that the condition is inherent in the tooth germs and is not due to external influences. Roentgenograms of the developing third molars confirm that the enamel is abnormal, even from the early stages of calcification, and the appearance on eruption is not due to resorption of enamel before eruption.

The cases of hypocalcification appear to bear a much closer resemblance to one another than do the cases of hypoplasia, and it appears that quite frequently the incisal and occlusal parts of the enamel are more severely affected. The considerable wear and staining that occur after eruption in these cases completely alter the clinical appearance of the teeth, and the extent to which they take place is dependent on the hardness of the enamel and the standard of the patient's oral hygiene. The oral hygiene is poor in Cases 3, 4 and 5 but quite good in Case 6. It is noticeable that the teeth in Case 6 are much less stained in comparison with those of the patient in Case 4 (a child of similar age). There is little doubt that poor oral hygiene is encouraged by the difficulty in cleaning the rough enamel surface and the general unpleasant appearance of the teeth.

A comparison of eruption times of the permanent teeth in these children shows that only in Case 1 is there any delay or failure. In all the other children, eruption is proceeding quite normally, irrespective of the condition of the enamel. The crowns of the unerupted first permanent molars in Case 1 appear to be undergoing resorption as can be seen in the roentgenograms (Fig. 4, *A* and *B*) taken two and one-half years apart. During this period there has been no sign of any movement of these molars toward the occlusal plane, but the premolars, on the other hand, have undergone active eruption with no signs of resorption. According to Weinmann, Svoboda, and Woods[7] the noneruption and resorption of the teeth in these cases are due to early degeneration of the reduced enamel epithelium (Nasmyth's membrane), the presence of which is said to be necessary to cause atrophy of the tissues blocking the path of eruption and to protect the tooth crown from resorption. In Case 2, however, the premolars are practically devoid of enamel and presumably of Nasmyth's membrane, and yet they erupted quite normally. Possibly the presence of a deciduous predecessor has some influence on eruption in these cases, since only the molars are unerupted in Case 1 and all teeth with deciduous predecessors are erupting. The molars in Case 2 erupted normally, but of course they are not very hypoplastic. In general, it would seem that although arrested eruption does occur in hypoplasia it is not a constant feature of the condition.

SUMMARY

Six cases of amelogenesis imperfecta, of which two show hypoplasia and four show hypocalcification of the enamel, have been described. Both these forms of amelogenesis imperfecta may occur with or without a family history,

and the deciduous dentition may or may not be affected. The low rate of caries incidence and the absence of any other ectodermal defects are to be noted. The effects of wear and staining from the mouth in hypocalcification have been described.

Some of the cases described show unusual individual variations within this classification; one case shows arrested eruption, and this has been discussed.

I am indebted to Mr. J. N. Ainsworth, M.C., for permission to publish details of Case 1 and to Mr. P. M. C. James for Cases 3 and 4. I also wish to thank the Radiographic and Photographic Departments of the Royal Dental Hospital of London, England, for the roentgenograms and photographs.

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SUBLINGUAL "RANULA," DERMOID CYST, AND LIPOMA

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THE origin of the term "ranula" is somewhat obscure. Its derivation is from the diminutive form of the Latin *rana* ("the frog"), and its usage is commonly explained on the basis of the expression "a frog in the throat," implying hoarseness, or possibly the resemblance of the translucent lining of the cyst to the belly of the frog. Recourse to a Latin dictionary gives another possible explanation, as the term *rana* was used by the Latin author Columella to describe a disease of the tongue of animals.

Whatever may be the origin of the term, its use frequently gives rise to a good deal of confusion, since different authors have used it to embrace rather different conditions.

Dorland's *American Illustrated Medical Dictionary*[1] defines "ranula" as "a cystic tumor beneath the tongue, due to obstruction and dilatation of the sublingual or submaxillary gland or of a mucous gland." It continues, however, by saying that a pancreatic ranula is "a retention cyst of the pancreatic duct." The little frog appears to have traveled a long way! Stones[2] states: "The term ranula is often loosely applied to all forms of cysts of the floor of the mouth, including thyroglossal cyst. Some writers limit the term to cysts arising from occlusion of the excretory duct of the submandibular gland and the ducts from the sublingual gland. In this text the term will be confined to cysts arising in connection with the sublingual gland and mucous glands of the floor of the mouth. They are of fairly rare occurrence." He later quotes and illustrates a case in which "the submaxillary duct may be seen coursing over it [the "ranula"] as an opaque strand."

Thoma[3] defines "ranula" as "a large mucous cyst found in the floor of the mouth." However, he says of its etiology: "Occlusion of the duct of any of the sublingual mucous or salivary glands may cause retention of secretion and cyst formation."

Fish[4] states that "ranula" is "a term applied to any retention cyst of the floor of the mouth generally due to inflammatory obstruction of sublingual ducts." He widens the scope of the term tremendously, however, by continuing:

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"similar swelling may be caused by a fragment of salivary calculus, the removal of which through an incision over the obstruction permits the free escape of both the calculus and the saliva and cures the condition. Wharton's duct is the one usually involved in ranula, but Stensen's duct may be similarly affected."

Miller[5] lists "ranula" separately from mucous retention cysts and states of ranula: "It generally involves the submaxillary glands . . . a stone is usually found blocking the duct of the affected gland."

Burket,[6] on the other hand, evidently does not consider that the term "ranula" should embrace cystic swellings of the submaxillary duct or gland, since he states: "Pressure on the salivary ducts from pathologic processes *of the surrounding tissues* [italics mine, A. B. M.] results at times in partial or complete blockage of salivary flow. A large ranula may prevent flow of saliva from Wharton's duct and give rise to secondary symptoms of the submaxillary gland."

It will be seen from the foregoing short résumé of opinions culled from current textbooks that while there is general agreement that the term "ranula" should not include all forms of swelling in the floor of the mouth, there is no similar agreement on whether cysts of the mucous and sublingual glands should alone be included or whether cysts of the submaxillary ducts and glands should be included in addition.

The three following examples, all with the diagnosis of "ranula," referred to me for operative treatment in the professorial oral surgery unit of the Birmingham General Hospital give further evidence of the miscellany of conditions that may be included in the term "ranula," a term that I venture to suggest is too vague to be useful and should be abolished.

CASE REPORTS

CASE 1.—The first patient referred with the diagnosis of "ranula" was a 36-year-old man, who complained of a swelling under the tongue that had first been noticed three months previously. His dental surgeon had inserted a needle and withdrawn some fluid, but the swelling had recurred and was now bigger. On examination, it was found that a cystic swelling was present on the left side, under the tongue; this swelling was fluctuant and the coloration bluish (Fig. 1). Under local anesthesia, the top of the cyst was excised and the edges lightly sutured to the margins of the base. Healing was uneventful, and there was no recurrence.

This was a mucous retention cyst and comes within the generally accepted definition of a "ranula."

CASE 2.—The second patient referred with the diagnosis of "ranula" was a 31-year-old woman. The history was a little vague, but, according to the patient, a swelling in her neck had been noticed when she was only 14 days old and she had had a steel plate applied externally in order to compress it. The swelling had vanished, but for the last few years she had noticed a painless swelling under the tongue. This had appeared to increase in size during the last few months, after extraction of some lower teeth. On examination, a round, smooth, painless swelling about the size of a large marble was seen in the floor of the mouth under the base of the tongue (Fig. 2). The swelling was soft but did not fluctuate,

Fig. 1.—Mucous retention cyst occurring under the tongue.

Fig. 2.—Dermoid cyst occurring under the tongue.

Fig. 3.—Dermoid cyst after removal from tissues beneath the tongue.

and it appeared to arise from deep tissues below the tongue. It was situated slightly to the right of the midline, and part of the right submaxillary duct could be seen running over the surface. Radiographs were negative.

The patient was admitted to the hospital. Under endotracheal general anesthesia, a sausage-shaped cyst with thick walls was exposed by a vertical incision below the tongue and anterior to the openings of the submaxillary salivary ducts. The base of the cyst was found well down in the tissues toward the hyoid, and in parts dissection was made more difficult by surrounding fibrosis. The cyst having been removed (Fig. 3), the wound was closed by deep and superficial sutures. On the following day there was slight distention of the right submaxillary salivary gland and along the line of the duct. One superficial suture was removed, and the fact that this had been obstructing the duct was shown by an immediate escape of saliva from it. Healing was subsequently uneventful. When the cyst was cut open, it was found to contain thick, white, caseous, inspissated material. This confirmed the diagnosis of dermoid cyst (Fig. 4).

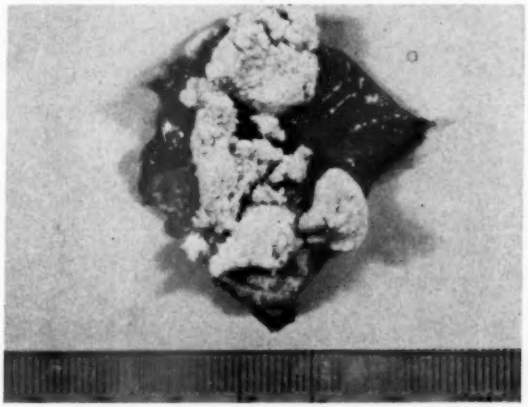

Fig. 4.—Dermoid cyst cut open to show caseous contents.

On biochemical examination, the white material was found to contain numerous cholesterol crystals, some epithelial debris, and occasional neutral fat globules. On histologic examination, the cyst lining was found to be formed from stratified squamous epithelium but no hairs or hair follicles were found (Fig. 5). In one area where the lining epithelium was absent a large congregation of giant cells was seen (Fig. 6).

CASE 3.—The third patient sent in with a diagnosis of "ranula" was a lady of 77 who complained of a painless swelling under the tongue which she had noticed for eighteen months. It had first been evident shortly after a loose tooth had been extracted and, according to the patient, had been slowly growing since. The old lady was not very clear about the history, and considerable cancerophobia was present. It is probable that the swelling had, in fact, been present for a longer time.

On examination, as soon as the patient opened her mouth, a large, soft swelling about 5 cm. in diameter, arising from the floor of the mouth under the tongue slightly to the left of the midline, protruded between the lips. The tongue was forced upward, speech was difficult, and the remaining lower incisor teeth, which were very loose, were displaced labially. The swelling was the same color as the mucosa, painless, slightly lobulated, and fluctuant (Fig. 7). The salivary glands appeared normal; no swelling of lymphatic nodes could be detected; and roentgenograms were negative. In view of the marked fluctuation

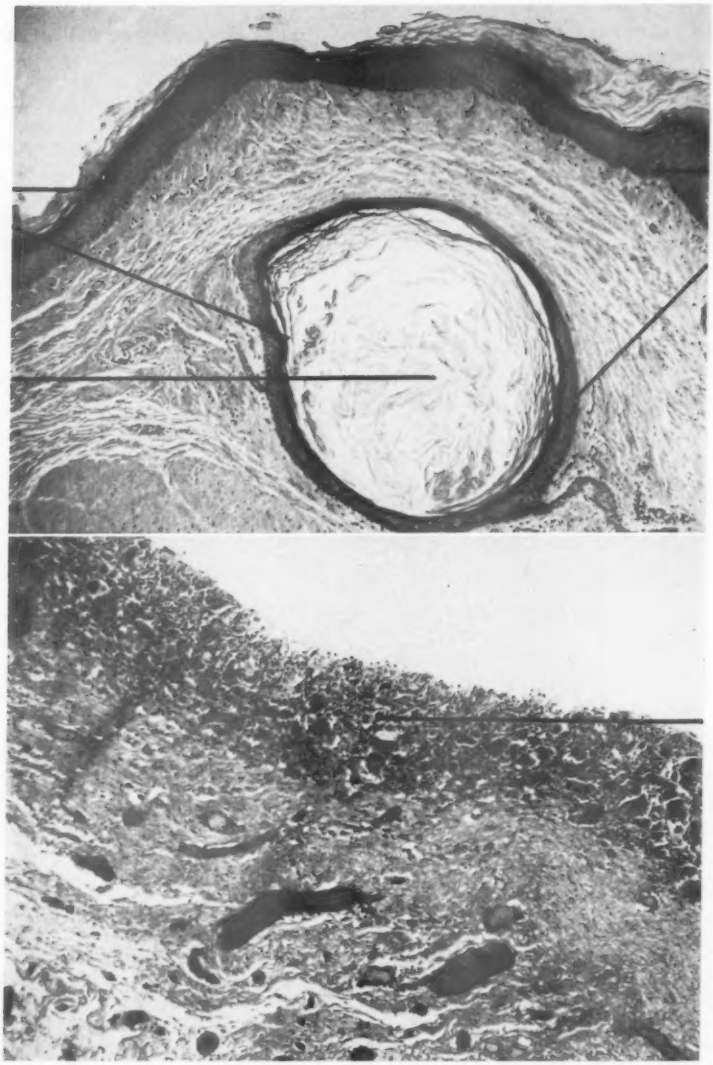

Fig. 5.—Photomicrograph of part of wall of dermoid cyst showing small invaginated portion of cyst (*A*) within the main cyst lining. *SE*, Squamous epithelium. *K*, Keratinized portion of epithelium. (Magnification, ×120.)

Fig. 6.—Photomicrograph of part of wall of dermoid cyst where the epithelial lining was absent showing large numbers of giant cells (*GC*). (Magnification, ×120.)

and transmission of light on transillumination, an attempt was made at aspiration under local anesthesia. When this failed, a small incision made for biopsy showed at once that the swelling was a lipoma.

Fig. 7.—Lipoma arising from under the tongue.

Fig. 8.—Lipoma after removal. The mucosa overlying the top half of the specimen was removed deliberately in order not to leave excess mucosa in the mouth.

In view of the age and rather nervous state of the patient, it was decided not to continue the operation under local anesthesia, and she was admitted to the hospital. Under endotracheal anesthesia, the mucosa was incised, the lipoma was shelled out in one piece, redundant mucous membrane was removed, and the incision was closed. The remaining loose lower incisors were extracted. Healing was uneventful, and section of the tumor (Fig. 8) confirmed the diagnosis of lipoma (Fig. 9).

DISCUSSION

Ranula.—While not particularly common, the cystic condition described in the first patient could not be described as rare. In this instance, though clearly a retention cyst, it was not clear whether its origin was from obstruction of a duct of the sublingual gland or of one of the glands of Blandin and Nuhn or whether it was caused merely by occlusion of the duct of a mucous gland.

The similarity in appearance and contents between this and other examples seen by me and those of the simple mucous cyst of the lip and cheek strongly suggests that many, if not all, cases of "ranula" are, in fact, sublingual mucous retention cysts.

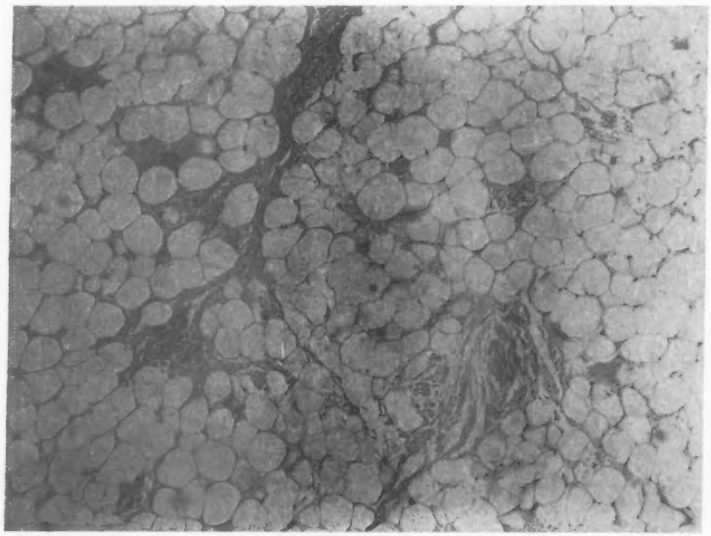

Fig. 9.—Photomicrograph of lipoma showing typical appearance of true lipoma. (Magnification, ×100.)

Sublingual Dermoid Cysts.—These are comparatively rare. Roser,[7] in 1859, first called attention to the fact that some so-called "ranulae" and cysts found at the base of the tongue were, in fact, epidermoid tumors.

In 1937 New and Erich,[8] reviewing the figures of dermoid cysts seen at the Mayo Clinic for the twenty-five years from 1910 to 1935, showed that of 1,495 such cysts only 103 (6.94 per cent) occurred in the head and neck, and of these only twenty-four (1.6 per cent) of the total were present in the floor of the mouth. Meyer,[9] in 1955, pointed out that so far less than 160 sublingual dermoid cysts had been reported in the literature and many of the earlier reports gave no histologic proof of the diagnosis. Distribution appeared to be equally divided between the sexes, and the patients seeking treatment appeared to range between 15 and 35 years of age.

There is general agreement that dermoid cysts in this position are derived from epithelial debris or rests included during the midline union of the first and second branchial arches. They arise usually in the midline and may become obvious either below the geniohyoid muscle (submental) or above the geniohyoid muscle (sublingual).

There is some disagreement as to whether sublingual dermoid cysts contain hair or whether hair follicles are present. Thus, Hamilton Bailey[10] states: "Sublingual dermoids seldom (if ever) contain hair." This view is supported by New and Erich. On the other hand, Meigs[11] gives evidence of hair being present in one or two such cysts, and Meyer[9] states that hair follicles were present in the walls of three out of four sublingual dermoid cysts examined.

Other authors who report cases (for example, Colp,[12] Schaefer and Rudder,[13] Musgrove,[14] Korchin,[15] Terracol,[16] and Kaskel and Schaffer[17]) do not give information on this point. In the present example no hairs or hair follicles were present.

Lipoma.—Lipomas may, of course, occur in any region where fatty tissue is normally present. They are said to be of congenital origin in some instances, but as so many occur in older persons it would appear unlikely that this view could be substantiated in the majority of cases.

While they are not uncommon in other parts of the body, they appear to be rare in the oral cavity. Geshickter,[18] reviewing 490 lipomas, found that only three occurred in the mouth. Of those occurring in the mouth, sublingual lipoma appears to be one of the rarest forms. Of three examples of intraoral lipoma collected by Bruce and Royer,[19] all were in the cheek or buccal fold attached to the gingiva. In other instances, such as those cited by Thoma,[20] they were found arising in the postzygomatic fossa or from the tongue itself, as also described by Giovanni[21] and others.

Thoma[20] does, however, describe and illustrate one case in which a lipoma was found arising from beneath the mucosa under the tongue, and one further example is described by Churchill.[22]

Of the three patients with the diagnosis of "ranula" sent to me for operative treatment, only the first fulfills the criteria of the generally accepted definition. If, however, the term is held to embrace all cystic swellings arising under the tongue, the dermoid cyst should also be included. It could even be argued, although perhaps more speciously, that from its position and appearance, if not its pathology, it would not be wrong to include lipoma.

The term "ranula" is, in fact, so vague, unscientific, and capable of varied definition that it would be better to abolish it. If we mean a sublingual mucous or salivary retention cyst, we should say so.

SUMMARY

Three patients seen with the diagnosis of "ranula" are described. One had a mucous retention cyst, another had a sublingual dermoid cyst, and the third had a large lipoma. Because of the comparative rarity of a dermoid cyst or

lipoma in this position, the literature and the pathology of these two conditions are discussed more fully. In view of the conflicting definitions of "ranula" given in different textbooks and by different authorities, it is suggested that the term "ranula" should be abolished in order to avoid confusion.

I would like to record my thanks to colleagues who first referred the patients to me for treatment, to Dr. E. A. Marsland who most kindly undertook the photomicrography for me, and to Mr. G. A. Johnson of the technical staff of the Department of Dental Pathology for his help with the photographing of the specimens.

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A DEVELOPMENTAL ANOMALY IN THE FORM OF AN OCCLUSAL TUBERCLE WITH CENTRAL CANAL WHICH SERVES AS THE PATHWAY OF INFECTION TO THE PULP AND PERIAPICAL REGION

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THE purpose of this article is to present our studies on a developmental anomaly observed in premolars (Fig. 1), in the form of an occlusal tubercle which serves as a pathway of infection to the pulp and to the periapical area. In 1955 Lau[1] reviewed the literature on this type of anomaly and reported fifty-two cases observed in Chinese persons, not only in the premolars but also in other teeth. He pointed out that in 1936 Yumikura and Yoshida, two

Fig. 1.—A photograph showing an intact occlusal tubercle in the upper left first premolar. (Courtesy of Dr. T. C. Lau.)

Japanese investigators, had described this anomaly as observed in Japanese persons. It was thought previously that Leong Ming Ong had been the first to report this anomaly in 1946; hence, one author[2] called this anomaly "Leong's premolar."

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The development of this occlusal tubercle seems to be the reverse of dens in dente. Tratman[3] claims that it is due to an "invagination" of the inner enamel epithelium into the enamel organ, followed by the dental papilla. We subscribe, however, to Lau's opinion that the occlusal tubercle is apparently caused by the evagination or "extrusion" of a part of the inner enamel epithelium into the stellate reticulum occurring in the early stages of tooth development.[1] The proliferated enamel epithelium influences the underlying dental papilla to differentiate into odontoblasts and form dentine; thus, the tubercle has an outer enamel covering and a core of dentine around a central canal which is continuous with the pulp.

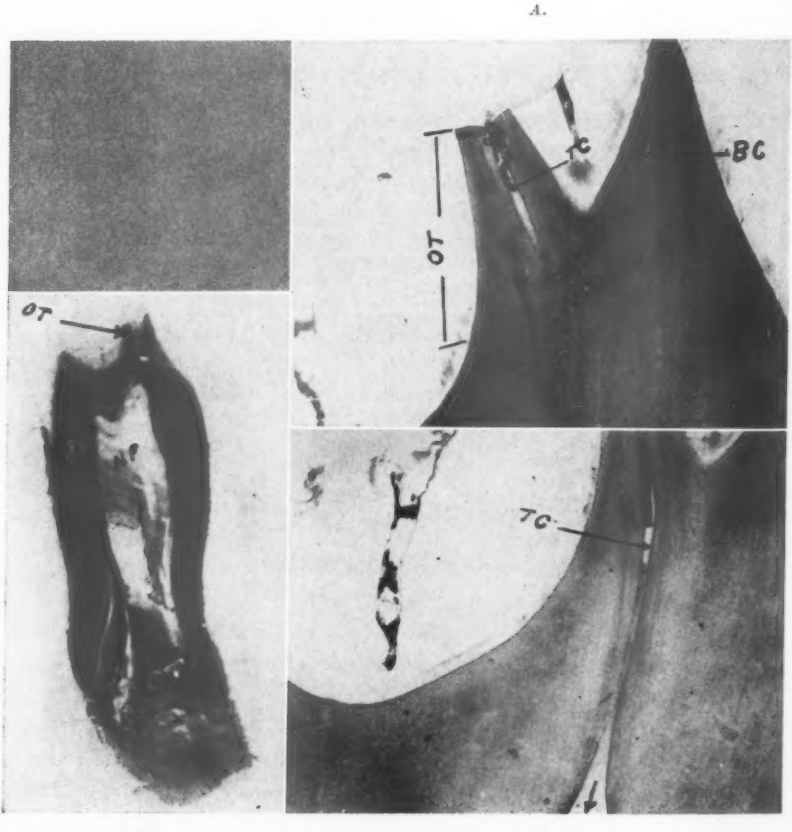


Fig. 2.—*A*, A photograph of a section showing a portion of the dentine of the occlusal tubercle (*OT*). The tubercle emerges from the triangular ridge of the buccal cusp. *D* Dentine; *NP*, necrotic pulp; *GT*, granulation tissue with abscess cavity at the center.

B, A photomicrograph of the occlusal tubercle (*OT*), showing a central canal (*TC*) opening to the outside. Note that the tubercle appears to be abraded and emerges from the triangular ridge of the buccal cusp (*BC*). (Courtesy of the *Philippines Medical-Dental Journal.*)

C, Another photomicrograph showing the central canal (*TC*) connecting with the pulp chamber indicated by the arrow.

In 1956[4] one of us (V. G. V.) observed a similar tooth anomaly associated with a chronic periapical abscess with extraoral sinus. The anomaly was discovered after the tooth, a noncarious lower premolar, was decalcified and sectioned for histologic study (Fig. 2, *A*). In Fig. 2, *B* and *C* are photomicrographs of the tubercle, showing the presence of a central canal. The plane of the section was not exactly parallel with the long axis of the tubercle; hence, a composite picture is presented to establish that the canal opens distally to the outside and proximally to the pulp chamber. The tubercle emerges from the triangular ridge of the buccal cusp, and its distal end appears to be abraded, thus exposing the canal to the outside. This article will deal with two additional cases which we have observed in Filipino patients within the period of one year. Both cases were discovered because they were associated with chronic periapical abscesses.

CASE REPORT

CASE 1.—A 22-year-old Filipino was admitted to the clinic complaining of pain in the region of the lower right second premolar, which was noncarious. The tooth was tender on percussion. An intraoral fistula exuding pus was present on the gingiva close to the

A.

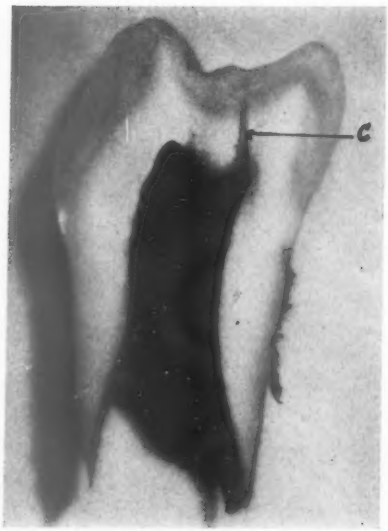
B.

Fig. 3.—*A*, A radiograph of the region of the lower right premolars. Note the second premolar which is noncarious, showing resorption of the root and rarefaction of the bone in the periapical region.

B, A photograph of the ground section of the second premolar. Note the evagination of a certain portion of the dentine into the enamel with a central canal (*C*) exposed to the outside and connected with the pulp chamber. *R*, A portion of the undeveloped roof of the pulp chamber; *D*, dentine; *E*, enamel.

tooth. A radiograph showed extensive resorption of the apical portion of the root of the tooth and rarefaction of the bone in the periapical region (Fig. 3, *A*). The tooth was extracted, and a ground section was made in a buccolingual plane (Fig. 3, *B*). A portion of the coronal dentine evaginated into the enamel with a central canal (marked *C*). The canal was exposed at the occlusal surface, evidently as a result of abrasion of the tooth. Proximally, the canal was connected with the pulp chamber. It may be noticed that a portion of the roof of the pulp chamber was present (marked *R*). It seems that the development of the roof of the pulp chamber had been interrupted by the formation of the proximal opening of the canal. The developmental anomaly appeared to be emerging from the triangular ridge of the lingual cusp of the premolar.

A.



B.

Fig. 4.—*A*, A radiograph of the region of the lower left premolars. Note the second premolar with resorption of the root and rarefaction of the bone in the periapical portion, including the mesial bony septum. Arrow points to an occlusal tubercle in the first premolar.

B, A photograph of the ground section of the second premolar showing a portion of the central canal (*C*) connecting with the pulp chamber. The distal end of the canal, supposedly opening to the outside, was removed during grinding of the specimen.

CASE 2.—An 18-year-old Filipino was admitted to the clinic complaining of recurrent pain, swelling, and pus discharge for the past two years in the region of the lower left second premolar. A radiograph showed resorption of the apical portion of the root of the

tooth and rarefaction of the bone in the periapical region, including a portion of the bony septum in the mesial side (Fig. 4, *A*). It may be noted that the second premolar was noncarious. The first premolar also showed an occlusal tubercle (indicated in the illustration by an arrow), with slight bone rarefaction at the periapical region. The second premolar was extracted, and a ground section was made (Fig. 4, *B*). It is regretted that the distal portion of the canal (marked *C*) was removed during grinding of the specimen. However, a greater portion of the canal opening to the pulp chamber was still present. This was a double bilateral case. In Fig. 5 a radiograph of the right side of the lower jaw shows the first premolar without caries but with some lesions in the periapical region. The second premolar also shows an occlusal tubercle (arrow).

Fig. 5.—A radiograph of the region of the lower right premolars of the same patient as in Fig. 4. Note the first premolar with a lesion in the periapical region. The second premolar also shows an occlusal tubercle indicated by an arrow.

COMMENT

The developmental anomaly in the form of an occlusal tubercle with central canal which has been observed among some Japanese and Chinese persons has also been observed among some Filipinos. The three cases reported here were associated with chronic periapical abscesses. This is also true of most cases reported by other authors. There is no doubt, therefore, that this type of anomaly has a clinical significance like most cases of den in dente frequently associated with infection of the pulp and, later, of the periapical region. It is evident that as soon as the occlusal tubercle comes in contact with the opposing teeth it is either fractured or abraded, thus exposing the central canal to the outside. This exposed canal, which could hardly be recognized by visual examination, becomes the portal of entry of the infection of the pulp and its complications. It is possible that many cases of periapical abscesses associated with noncarious teeth, the etiology of which has been frequently ascribed to past history of trauma inflicted on the tooth to hematogenous infection, may be due to this type of developmental anomaly which appears to be the reverse of dens in dente. It may be mentioned that the first case reported in 1956 was discovered only after the noncarious tooth was sectioned out of curiosity. The last two cases of chronic periapical abscesses associated with noncarious teeth were discovered as being due to this type of anomaly only because of past experience and after the teeth were sectioned for histologic study. It is difficult to recognize this anomaly if the occlusal surface of the tooth has already been abraded.

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LIPOMA OF ORAL CAVITY

Report of Seven Cases

H. E. Simpson, M.D.S., Newcastle upon Tyne, England

LIPOMA of the oral cavity is uncommon. By comparison, in the period during which the seven cases reported in this article were collected fifty cases of carcinoma and 110 cases of fibroma and fibrous epulis were observed.

The tumor consists of a mass of fat cells and is usually divided by fibrous septa into irregular-sized lobules. It elevates the mucosa and is often pedunculated. Any part of the mouth where there is normally fatty tissue may give rise to a lipoma (Figs. 1, 2, and 3). The lower lip and adjacent parts of the cheek are common sites. Usually there is only one tumor, but bilateral cases affecting the tongue or pharynx have occurred.[5]

If the overlying tissue is thin, the yellow fat can be seen through the mucosa. This, together with the soft, fluctuant nature of the tumor, makes the clinical diagnosis simple. If the color is not evident, it may be impossible to distinguish, when sessile, from a deeply placed mucous retention cyst or, when pedunculated, from a soft fibroma.

The history is variable, partly no doubt because the condition may not be observed until speech and mastication are affected. Congenital tumors have been

Fig. 1.—Lipoma of lip.

From the Department of Oral Pathology, King's College, Newcastle upon Tyne, England.

described in other parts of the body.[8] In the cases reported here the history varied from three weeks to fifteen years.

Lipomas in other parts of the body may reach a considerable size before treatment is sought, but in the mouth they are usually relatively small (for

Fig. 2.—Lipoma of cheek.

Fig. 3.—Lipoma of floor of mouth.

example, 3.5 by 3 cm. in Turner's[6] case and 1.3 by 1.1 cm. in the case reported by Dixon and Ziskind[2]). Paul,[4] however, reported a 6 ounce lipoma of the tongue, the weight and size of a large orange.

On operation, the oral tumor usually is easily shelled out and its nature becomes evident from its color and consistency (Fig. 4). On being incised, small globules of fat exude from the cut surface.

Histologically there is a rather thin but complete capsule and, generally there are septa of fibrous tissue (Fig. 5). The amount of fibrous tissue is variable and is sometimes sufficient to warrant the use of the term *fibrolipoma.*

Fig. 4.—Operative specimen from lipoma of lip shown in Fig. 1.

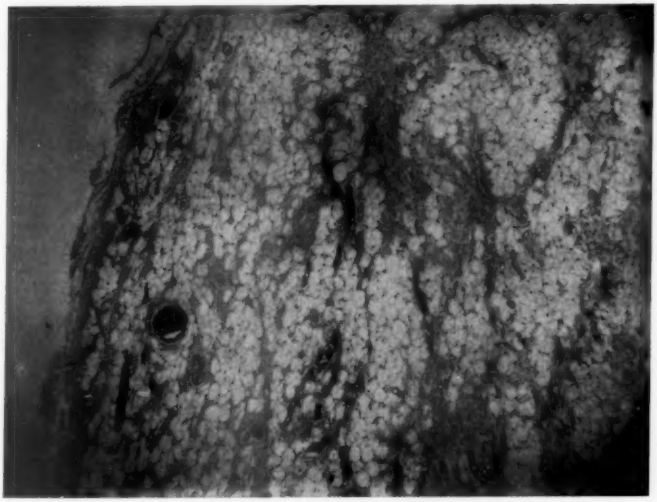

Fig. 5.—Photomicrograph of specimen shown in Fig. 4.

Lipoma is one of the most innocent of tumors, but on rare occasions a malignant form (liposarcoma) may be encountered.[1] According to Ward and Hendrick,[7] the deeper-placed tumors which occur in various parts of the body are less well encapsulated and more prone to become malignant.

During the past five and one-half years, seven cases of lipoma have been diagnosed and histologically examined in the Newcastle upon Tyne Dental Hospital. Six have been in middle aged or elderly patients but the age at which the

tumors first appeared must generally have been much less. In four cases the tumors were in situations on the lower lip or cheek where trauma could have been a factor in their formation. In this connection, Muir[3] stated that there appeared to be a relationship between pressure and the situation of skin lipomas.

TABLE I

AGE	SEX	SITE	OBSERVED DURATION	PEDUNCULATED OR SESSILE	GREATEST DIAMETER (CM.)
69	M	Lower lip	10 years	Pedunculated	2
47	M	Cheek	15 years	Pedunculated	2
32	F	Cheek	Not observed 7 years previously	Sessile	2
74	M	Cheek	Unknown	Pedunculated	1.5
48	F	Floor of mouth	15 years	Sessile	1
75	F	Cheek	3 weeks	Sessile	2.5
56	F	Lower lip	Unknown	Sessile	3

The details of the seven cases are presented in Table I. The fact that the tumors were pedunculated in all the male patients and sessile in all the female patients can hardly be considered significant.

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ENDODONTICS

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BACTERIOLOGIC EVALUATION OF THE GLASS BEAD STERILIZER FOR ENDODONTICS

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FLAHERTY[3] suggested molten metal as a potential sterilizing agent for use in the dental office. Boak[1] concluded that a five-second exposure of metal instruments contaminated with vegetative and sporulated organisms in molten metal was an effective means of sterilization; swabs with vegetative organisms required five seconds, but a ten-second exposure was required to destroy spores on cotton swabs. He used *Streptococcus viridans, Streptococcus hemolyticus, Staphylococcus sp.,* and *Bacillus subtilis.* No sterilizing temperatures were recorded. Stewart and Williams[6, 7] noted that a two-second exposure in molten metal repressed growth of organisms on contaminated metal instruments, but absorbent points and cotton pellets required five- and ten-second exposures, respectively, to effect sterilization. The spores of *Bacillus globigii,* carried on metal instruments, required a five-second exposure. Temperature ranges of 218 to 226° C. and 260 to 280° C. were used with the following organisms: *Staphylococcus aureus, Bacillus subtilis, Bacillus globigii,* a yeast, *Clostridium welchii,* and *Clostridium sporogenes.* Findlay[2] reported destruction of vegetative organisms on metal instruments after nine seconds' exposure at 420° F.; *Bacillus subtilis* required nine seconds' exposure at 520° F. Absorbent points, contaminated with vegetative organisms, required ten seconds' exposure at 420° F.; those contaminated with *Bacillus subtilis* needed seventeen seconds' exposure at 520° F. With cotton pellets, vegetative organisms were innocuous after an eleven-second exposure and sporulated organisms were innocuous after exposure for twenty-four seconds at 520° F. The organisms used were *Staphylococcus aureus, Streptococcus viridans, Streptococcus hemolyticus, Bacterium coli, Proteus vulgaris,* and *Bacillus subtilis.* Grossman,[4] in a study of rapid sterilization methods, used a sterilizer with glass beads in contact with the test instruments in lieu of molten metal mixtures. He noted that the technique required small beads "like fine grains of sand"

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for effective heat transfer, a temperature range of 225 to 250° C., and a temperature differential of 30° C. within the sterilizer. Although neither organisms nor time intervals were specified, Grossman suggested that the duration of heat exposure should be equivalent to the conventional times used for molten metal sterilization. A temperature analysis of thermostatically controlled root canal sterilizers was reported by Oliet, Sorin, and Brown,[5] who determined that a definite temperature gradient was exhibited between the base and surface and the outer circumference and center of the well of all sterilizers.

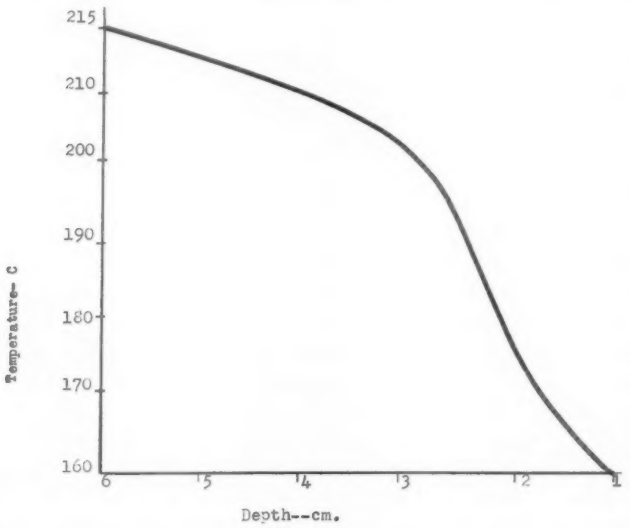
Manufacturers' literature* suggested that with 1 mm. solid glass spheres and a temperature of 450° F., a three- to five-second exposure for metal broaches and a ten-second exposure for paper or cotton material was required for sterilization of contaminated materials.

MATERIALS AND METHODS

A commercial glass bead sterilizer† was used in the following study.

Sterilizer temperatures were measured with a mercury-filled thermometer. Temperature strata as a function of immersion depth were observed and are recorded in Table I with emergent stem corrections applied to the data.*

TABLE I. TEMPERATURE VARIATION AS A FUNCTION OF IMMERSION DEPTH



Because of the temperature strata, all broaches and paper points were immersed to the bottom of the sterilizer, to ensure a more constant heat exposure of the test instruments.

*Electro-ball sterilizer, Union Broach Co., Inc., New York, New York.
†Emergent stem correction is 11.5° C.

Efficiency of heat sterilization by the ball sterilizer was determined on an "all-or-none" basis. Dental broaches, paper points,* and associated equipment were autoclaved at 121° C. for twenty minutes and contaminated by seizing the broaches or points with sterile forceps and immersing them in a twenty-four-hour culture of test organisms. They were removed after thirty seconds' exposure and placed upon a sterile paper filter in a covered Petri dish to remove excess moisture. Then the contaminated dental materials were removed with forceps from the Petri plates and held among the glass beads in the ball sterilizer for varying times; they were removed, dropped into recovery broth, and incubated at 37° C., and the results were recorded after seventy-two hours. The results of these experiments are shown in Tables II and III. Recovery broth used in these experiments was either trypticase† soy or veal infusion.‡ In all cases, experimentally contaminated broaches and paper points grew well in both types of recovery broth.

Test organisms used in this study were *Streptococcus fecalis* (an enterococcus), *Staphylococcus albus, Escherichia coli,* and *Monilia albicans.*

RESULTS AND DISCUSSION

The commercial ball sterilizer used in these experiments displayed approximately a 52° C. temperature variation (corrected for emergent stem) between the bottom of the unit and the surface of the glass balls, as charted in Table I. This variation was surprising but not unexpected, since the construction of the unit placed all heating coils in the bottom with a modified Dewar flask as the container. Irrespective of ambient temperatures, the highest temperatures noted in this study were between 210 and 215° C. at the floor of the unit. Standardization of the technique resulted in the introduction of all test substances to the bottom of the unit, a 6 cm. immersion depth. At this position daily variations in temperatures were minimized to an increment of ±4° C.

Table II shows the time exposure required to sterilize contaminated metal broaches. With *E. coli* as the test organism, broaches were sterile with a one-second exposure to 211° C. In a few instances, a two- or three-second exposure was required to effect sterilization, in accord with the two-second exposure designated by Stewart and Williams. *Staphylococcus albus* was usually destroyed by a two-second exposure, although individual experiments required, on occasion, a four-second exposure before no growth would occur in the recovery broth. *Streptococcus fecalis* exhibited the most erratic results, for out of seventeen runs only eight showed no skips. The misses ranged from one to nine seconds, with the average being six seconds. Thus, for effective sterilization of broaches, a nine-second exposure is required; this demonstrates the heat-resistant property of this organism. *Monilia albicans* was the most heat-sensitive organism used, since there were no skips recorded and only a one-second immersion in the glass bead sterilizer was required to obtain negative results in the recovery broth.

*Johnson & Johnson, New Brunswick, New Jersey, manufacturers.
†Baltimore Biological Laboratories, Inc., Baltimore, Maryland.
‡Difco Laboratories, Detroit, Michigan.

TABLE II. RESPONSE OF MICROORGANISMS ON METAL BROACH CARRIERS TO STERILIZING TIMES

ORGANISM	TIME (SECONDS)											CULTURE MEDIUM	TEMPERATURE (DEGREES C.)	
	C*	1	2	3	4	5	6	7	8	9	10		STERILIZING	ROOM
Escherichia coli	+	–	–	+	–	–	–	–	–			T. soy	215	24
	+	–	–	–	–	–						T. soy	215	24
	+	+	+	–	–	–						T. soy	210	21
	+	+	–	–	–	–						T. soy	210	21
	+	–	–	–	–	–						V. inf.	213	23
	+	–	–	–	–	–						V. inf.	213	23
Staphylococcus albus	+	–	–	–	–	–	–	–	–			T. soy	215	24
	+	–	–	–	–	–	–	–	–			T. soy	215	24
	+	–	+	+	–	–						T. soy	211	25
	+	–	–	–	–	–						T. soy	211	25
	+	+	–	–	–	–						T. soy	210	21
	+	–	–	–	–	–						T. soy	210	21
	+	+	–	–	–	–						V. inf.	213	23
	+	–	–	–	–	–						V. inf.	213	23
Streptococcus fecalis	+	–	+	+	–	+						T. soy	212	25
	+	+	+	+	+	+						T. soy	212	25
	+	+	–	+	+	+	–	+	–			T. soy	210	24
	+	+	+	+	+	+	+	+	+	+	–	T. soy	215	24
	+	+	+	+	+	+	+	+	+	+	–	T. soy	215	24
	+	+	+	+	+	+	+	+	–	+	–	T. soy	215	24
	+	+	+	+	+	+	+	+	+	–	–	T. soy	211	22
	+	+	+	+	+	+	+	+	+	+	–	T. soy	211	22
	+	+	+	+	+	+	+	+	–	+	–	T. soy	211	22
	+	+	+	+	–	–	+	+	–	+	–	T. soy	211	22
	+	+	+	+	–	–	–	–	+	–	–	T. soy	213	24
	+	+	+	+	–	–	–	–	–	–	–	T. soy	213	24
	+	+	+	–	+	–	+	–	+	–	–	T. soy	213	24
	+	+	+	–	+	–	+	+	–	+	–	T. soy	210	24
	+	+	+	+	+	+	+	–	–	–	–	T. soy	214	23
	+	+	+	+	+	+	–	–	–	–	–	V. inf.	214	23
	+	+	–	–	+	+	–	–	–	–	–	V. inf.	214	23
Monilia albicans	+	–	–	–	–	–						T. soy	210	24
	+	–	–	–	–	–						T. soy	210	24
	+	–	–	–	–	–						T. soy	210	24
	+	–	–	–	–	–						T. soy	212	23
	+	–	–	–	–	–						T. soy	212	23

Key to Abbreviations:
T. soy = Trypticase soy broth.
V. inf. = Veal infusion broth.
*C = Control.

Table III shows the time required for sterilization of contaminated paper points. For all test organisms, a one-second immersion was sufficient for effective sterilization; however, this was contrary to the five- to ten-second exposure noted by other investigators.

CONCLUSION

Design changes to minimize the temperature variation would suggest heating coils about the entire depth of the unit so as to maintain a high uniform temperature, even at the 3 cm. depth which is equal to the length of an average paper point. This unit was so constructed that it had a funnel-shaped opening onto which, the manufacturer claims, a disk containing absorbent points and broaches may be placed, thus effecting partial sterilization. However,

TABLE III. RESPONSE OF MICROORGANISMS ON PAPER POINT CARRIERS TO STERILIZATION TIMES

ORGANISM	TIME (SECONDS)								TEMPERATURE (DEGREES C.)	
	C*	1	2	3	4	5	6	7	8	STERILIZING	ROOM
Escherichia coli	+	–	–	–	–	–	–	–	–	213	23
	+	–	–	–	–	–	–	–	–	213	23
	+	–	–	–	–	–				210	22
	+	–	–	–	–	–				210	22
Staphylococcus albus	+	–	–	–	–	–	–	–	–	212	22
	+	–	–	–	–	–	–	–	–	212	22
	+	–	–	–	–	–				211	23
	+	–	–	–	–	–				211	23
Streptococcus fecalis	+	–	–	–	–	–	–	–	–	213	22
	+	–	–	–	–	–	–	–	–	213	22
	+	–	–	–	–	–	–	–	–	215	24
	+	–	–	–	–	–	–	–	–	215	24
	+	–	–	–	–	–	–	–	–	215	24
	+	–	–	–	–	–	–	–	–	210	21
Monilia albicans	+	–	–	–	–	–	–	–	–	210	22
	+	–	–	–	–	–	–	–	–	210	24
	+	–	–	–	–	–				213	24
	+	–	–	–	–	–				213	22

Trypticase soy broth was used as the culture medium in this experiment.
*C = Control.

one disadvantage presented is that in order to insert an instrument into the glass beads it is necessary first to remove the dish, always with the possibility of dropping it. Also this sterilizer facilitates heat loss by nature of the large aperture. These two disadvantages can be corrected by constricting the opening to ¾ inch, instead of 1½ inches, and by inserting a small test tube into the glass beads. In this way it is possible to place instruments into the tube and partially sterilize them without having to remove them to insert instruments into the glass beads.

For effective sterilization of broaches contaminated with *Escherichia coli* a one-second exposure was required; *Staphylococcus albus* required two seconds, *Monilia albicans* required one second, and *Streptococcus fecalis* required nine seconds. Thus, for sterile broaches, a nine-second immersion in the glass bead sterilizer is required. Absorbent points inoculated with all organisms used in this study were innocuous after a one-second exposure. Therefore, the time needed for effective sterilization of all points is one second.

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RELATIONSHIP OF PAIN TO PULP DAMAGE

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THE following case is presented to illustrate the point that very deep cavity preparations with extensive pulpal damage, and even small pulp exposures under amalgam fillings, do not necessarily cause postoperative pain.

A 36-year-old white man was having full-mouth extractions prior to the construction of dentures. An occlusal cavity preparation in a noncarious lower first premolar was made with a steel burr No. 37 at 3,000 r.p.m. The preparation was made quite deep in order to assess the response of the pulp to the filling material, which in this case was amalgam.

After the amalgam was inserted, the patient was dismissed for eight days. At the time of his return, the patient was carefully questioned as to pain from biting pressure,

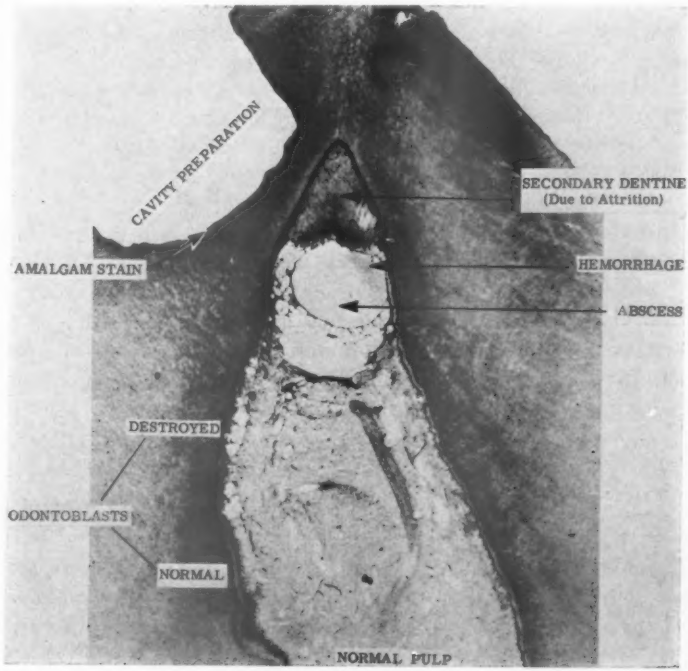

Fig. 1.—Experimental cavity preparation.

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thermal changes, or any other cause. His considered answer was that he had experienced no pain at all. The tooth was extracted, and after the usual processing procedures histologic sections were made. Microscopic examination (Fig. 1) revealed that the cavity preparation had been made very close to a pulp exposure, with extensive damage to the pulpal tissue resulting. There were hemorrhage and abscess formation within the pulp. The pulp below this area was not inflamed. The damaged area showed some signs of being walled off after only eight days. From the histologic picture, one would naturally *assume* that the patient was in constant pain. The fact was, however, that the patient had experienced no pain at all. In fact, he was not quite sure which tooth had been operated on!

Many similar cases were encountered during the course of this investigation, leading me to re-examine the question as to whether or not pain is always an indication of pulpal damage. It is not!

This case re-emphasizes the fact that the analysis of the effects of dental materials on the pulp must be based on careful histologic observations and not on clinical observations alone. It also supports the hypothesis that the odontoblast is the pain-receptor cell in the pulp and that death and destruction of this layer result in a failure of the pulp to receive painful stimuli. The conclusion drawn from this brief study is that absence of pain may indicate a healthy normal pulp or the death of the odontoblastic layer, without inflammation in the body of the pulp.

RESEARCH

TISSUE REPAIR IN INTRA-ALVEOLAR ROOT FRACTURES

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THE healing of intra-alveolar root fractures has long been a subject of interest to the oral pathologist, but opportunities for the examination of such fractures are seldom offered to him. Fractured teeth are frequently extracted too soon after injury to be of value for pathologic investigation, or else, if vital, they are retained until healing has taken place and their removal is no longer necessary unless for some other cause. In this way the number of fractured teeth available to the pathologist for histologic examination, especially during the early or intermediate stages of healing, is relatively small.

On the other hand, healed root fractures have been studied in both ground and decalcified sections of extracted teeth,[9, 12, 16, 17, 20, 24] but these represent only the end or final results of the healing process. Separate fragments of fractured roots extracted at varying intervals following injury have also been studied microscopically,[1, 12-15, 19] but a proper interpretation of the mechanisms of repair cannot be gained from such dissevered fragments.

For an adequate investigation of the repair processes, the root fragments must be studied in their proper relationship to each other and to their supporting tissues. For this purpose, an extensive surgical resection of the alveolar bone surrounding the fractured tooth is necessary but, of course, the occasion for undertaking such a procedure seldom arises in the human patient. Several specimens of fractured teeth have been obtained in this manner, however, and their subsequent histologic examination has yielded valuable information concerning the healing processes.[5, 6, 10, 18, 23]

In spite of the difficulty in obtaining specimens of fractured teeth, it is surprising to find that more use has not been made of the opportunities available during autopsy examination to resect relatively large sections of injured jaws. Only two instances appear to have been recorded in which specimens of fractured teeth were obtained in this manner, and in each of these the discovery of the fractured tooth was accidental.[7, 11] Further information regarding the

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healing mechanisms of root fractures has, of course, been gained from experimental studies of root fractures in rats[22] and dogs,[8] but only in the latter animal do the repair processes appear to conform with those described in the human being.

In the present study the histologic findings in two fractured maxillary incisor teeth are reported. These teeth were removed surgically together with their surrounding alveolar bone only six months after injury, while the repair processes were still active and before any union of the hard tissues of the teeth had taken place. Their early removal in this manner was undertaken so as to facilitate the subsequent orthodontic treatment for correction of a very marked superior protrusion.

CASE REPORT

A boy, aged 11 years 2 months, who was referred to hospital for orthodontic treatment, complained of discomfort and difficulty in mastication due to the excessive mobility of his maxillary central incisors. These teeth had been injured in a cycling accident six months previously, following which a cap splint had been fitted to the anterior maxillary teeth for three months.

On examination, both maxillary central incisors were found to be extremely loose and slightly tender to pressure, but the crowns were intact and the teeth reacted to vitality tests. Radiographic examination revealed transverse fractures of the middle thirds of the roots of both central incisors, and there appeared to be a considerable amount of separation of the root fragments (Fig. 1).

Fig. 1.—Radiograph of the maxillary central incisors showing the transverse fractures of the roots.

In view of the gross overcrowding of the maxillary and mandibular anterior teeth associated with a marked superior protusion, it was decided to resect the anterior part of the maxilla bearing the two fractured teeth. These teeth were therefore removed surgically en bloc with the supporting alveolar bone, and as soon as superficial healing of the soft tissues had taken place orthodontic treatment for approximation of the maxillary lateral incisors was commenced. The resected block of bone containing the two fractured teeth was submitted for histologic examination and serial sections were prepared of the decalcified specimen.

Histologic Findings.—

Right maxillary central incisor: The line of fracture in the right maxillary incisor was found to pass horizontally through the middle third of the tooth (Fig. 2). On the lingual aspect of the root the fracture appeared as a narrow irregular fissure in the dentine, but toward

Fig. 2.—Labiolingual section of the right maxillary central incisor showing the fracture of the root. (Hematoxylin and eosin stain. Magnification, ×7; reduced ¼.)

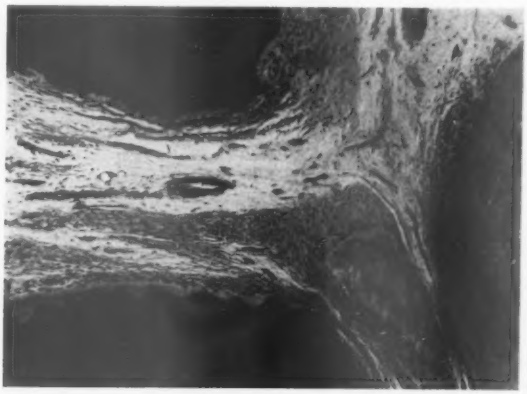
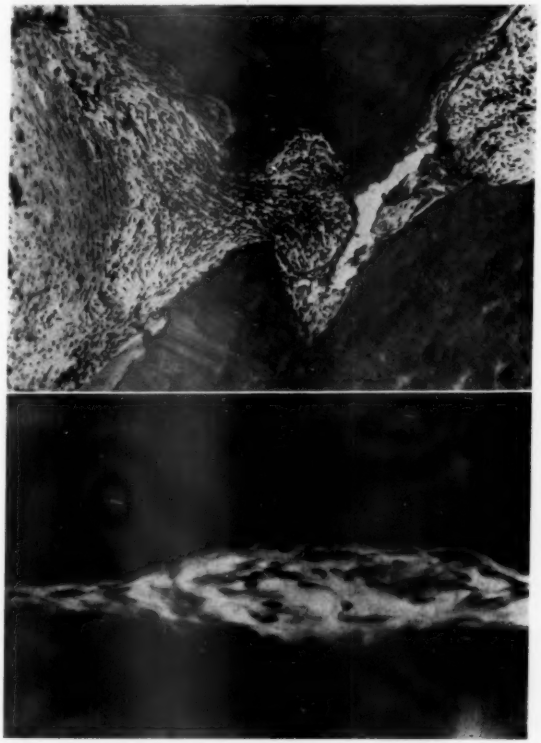


Fig. 3.—Labial extremity of the fracture in the right maxillary incisor. New bone formation is seen in the periodontal membrane, right. (Hematoxylin and eosin stain. Magnification, ×65; reduced ¼.)

the labial side of the root the space between the fractured root surfaces became wider and was filled by a broad band of well-vascularized fibrous connective tissue. This fibrous tissue was continuous with the connective tissue of the periodontal membrane peripherally and with

the pulpal tissue centrally (Fig. 3). Extensive osteoclastic resorption of the opposing fractured dentine surfaces had taken place along this part of the fracture line, but relatively little resorption of the fractured surfaces had, as yet, taken place toward the lingual extremity of the fracture. Here the narrow fissurelike space between the root surfaces was filled with spindle-shaped cells resembling connective tissue fibroblasts (Fig. 4, *A*). These cells appeared to have invaded the fracture line from both the periodontal and pulpal surfaces and were closely applied to the smooth surface of the fractured dentine. They even penetrated some very fine lateral cracks which ran at right angles into the dentine from the main fracture line, and some of these cracks were so fine that no more than one or two cells were sufficient to occlude them (Fig. 4, *B*).

A.



B.

Fig. 4.—*A*, Lingual extremity of the fracture in the right maxillary incisor showing invasion of the fracture line by fibrous tissue but little resorption of the fractured dentine. (Hematoxylin and eosin stain. Magnification, ×65; reduced ¼.)
B, Higher magnification showing cells invading a narrow crack in the dentine. (Hematoxylin and eosin stain. Magnification, ×475; reduced ¼.)

Within the pulp chamber the pulp was replaced by densely packed, elongated cells arranged with their long axes parallel with the long axis of the tooth. These cells resembled ordinary connective tissue cells or fibroblasts and were supported by a rich vascular capillary network. Their main source of blood supply, however, was still derived from the apical vessels, although a free vascular anastomosis existed with the vessels of the periodontal membrane through the fracture line.

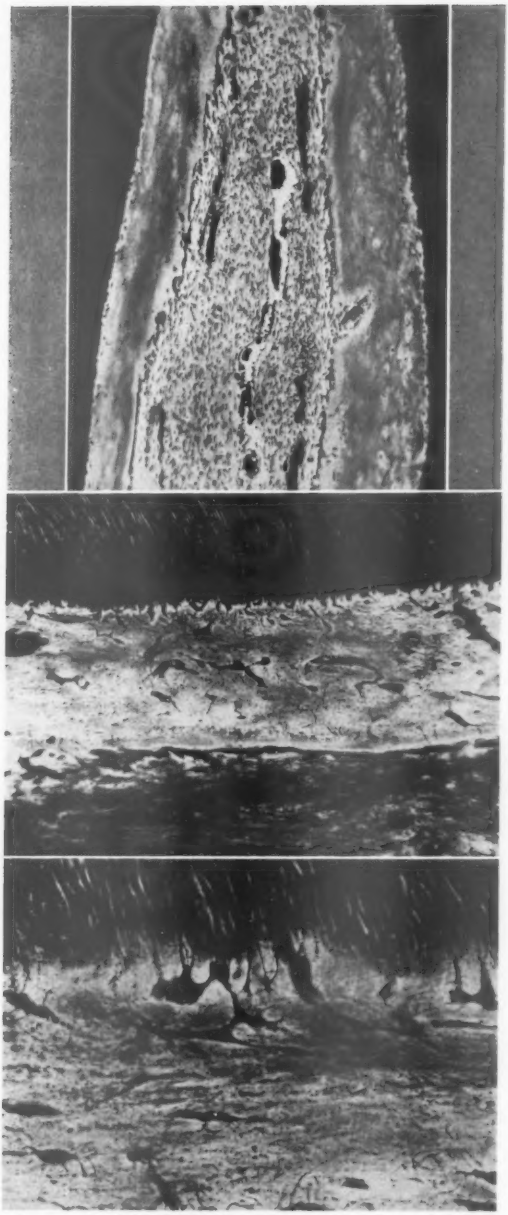


Fig. 5.—*A*, Cellular calcified layer lining the walls of the pulp chamber in the right maxillary incisor and replacement of the pulp by fibrous connective tissue. (Hemotoxylin and eosin stain. Magnification, ×90; reduced 3/10.)

B, Irregular nature of the cell inclusions in the calcified tissue lining the pulp chamber of the right maxillary incisor. (Picrothionine stain. Magnification, ×90; reduced 3/10.)

C, Cell inclusions close to the primary dentine showing canalicular connections with some of the dentine tubules. (Hematoxylin and eosin stain. Magnification, ×475; reduced 3/10.)

The odontoblast layer was absent from the pulp and instead of the normal predentine zone the pulp chamber was lined by a thick layer of calcified tissue containing numerous and irregular cell inclusions (Fig. 5, *A*). This layer of calcified tissue was directly continuous with the primary dentine, and its matrix appeared identical with that of the dentine. The cell inclusions were irregularly distributed throughout this matrix and were contained in small lacunae with two, three or more cells frequently occupying the same lacuna. Occasionally, some fine capillaries also entered the developing matrix and were found to terminate within the lacunae among the cell inclusions.

Stained by the picrothionine method, the outline of the lacunae and the canalicular pattern of this tissue was more easily studied (Fig. 5, *B*). The canaliculi were found to be extremely coarse and irregular and ramified from the lacunae for a varying distance into the surrounding matrix, sometimes joining with those from an adjacent lacuna. It was assumed that the canaliculi contained the protoplasmic processes of the cell inclusions, and those bordering the original primary dentine appeared to form direct connections with the free ends of some of the dentine tubules (Fig. 5, *C*).

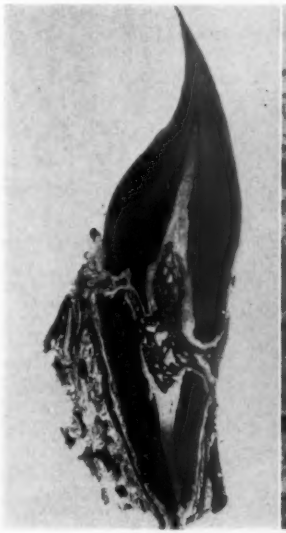


Fig. 6.

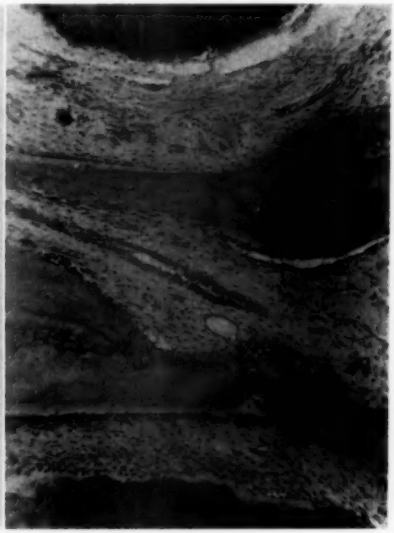


Fig. 7.

Fig. 6.—Labiolingual section of the left maxillary central incisor showing separation of the root fragments by a bridge of bone. (Hematoxylin and eosin stain. Magnification, ×7; reduced ¼.)

Fig. 7.—Well-formed lamellar bone between the fractured root ends of the left maxillary incisor. (Hematoxylin and eosin stain. Magnification, ×65; reduced ¼.)

Toward the apical portion of the root canal the predentine zone present at the time of injury could still be distinguished as a paler-staining zone between the original primary dentine and this newly apposed cellular calcified layer. Here, however, the cellular layer was much narrower than in the coronal portion of the pulp chamber, and new tubular dentine was beginning to form on the pulpal surface.

Surrounding this tooth the periodontal membrane was of uniform width, but the fibers appeared to have lost their definite lines of orientation. Opposite the fracture line, on the labial aspect of the tooth, a small spur of bone extended from the alveolar plate for a short distance into the connective tissue between the root fragments, but at no point did this bony

projection come into direct contact with the tooth substance (Figs. 2 and 3). The blood vessels in the periodontal membrane on the lingual aspect were widely dilated, a factor possibly exaggerated by the surgical manipulation of the specimen during its removal, and in the surrounding alveolar bone both osteoblastic and osteoclastic activity was much in evidence.

Left maxillary central incisor: In a labiolingual section of the left maxillary central incisor the line of fracture was found to pass obliquely through the middle third of the root (Fig. 6). In this tooth, however, the fractured portions of the root were completely separated from each other by a plate of bone which was continuous around its periphery with the alveolar bone of the socket wall. At its center this bony plate extended for some distance into the opened coronal and apical portions of the pulp chamber, but it did not form any direct union or ankylosis with the substance of the tooth. The bone consisted of well-formed lamellae, and the broad-surface osteoid seams indicated its rapid rate of growth (Fig. 7).

The dentine bordering the fracture line had undergone extensive osteoclastic resorption, and this had also accounted for a considerable excavation of the adjacent portion of the pulp chamber (Fig. 6). Elsewhere, however, the pulp chamber was lined with a layer of cellular calcified tissue identical with that described in the right incisor tooth. As in the latter tooth, the formation of this type of tissue appeared to be active in the coronal portion of the pulp chamber, whereas a modified form of tubular dentine formation had already commenced in the more apical part.

Fig. 8.—Partial resorption and fracture of the apex of the left maxillary incisor. (Hematoxylin and eosin stain. Magnification, ×30; reduced ¼.)

The pulp tissue was completely replaced by cellular fibrous connective tissue, and this in turn was continuous across the fracture line with the connective tissue of the periodontal membrane. The apical vessels and nerves were still intact, but there had been a small fracture of the extreme apex of the root of this tooth (Fig. 8). This may have been responsible for the resorption of part of the apex, but all the resorbed surfaces had been covered by a newly apposed layer of cellular cementum. The periodontal membrane contained many widely dilated vascular channels, but the usual orientation of its fibers was lacking. In the supporting alveolar bone, osteoblastic activity appeared to predominate.

DISCUSSION

Following traumatic destruction of any tissue, the repair process is initially concerned with the organization of blood clot and removal of the damaged tissues. This is followed by regeneration of the injured tissue or its replacement by a substitute or scar tissue. The appearance of the teeth studied in this report, therefore, represents an intermediate stage in the healing process, as the initial stage has been completed but the reparative processes are still active and the end result of healing cannot, as yet, be foreseen.

It is clear from the histologic findings that the response to injury has been similar in each tooth, except for the formation of a bridge of bone between the fractured portions of the left incisor. This bone has formed, presumably, as a result of metaplasia in the connective tissue separating the fractured fragments and, although the root fragments in the right incisor are in much closer apposition than those of the left incisor, there is, nevertheless, a small spur of new bone beginning to form in the connective tissue filling the labial extremity of the fracture line in this tooth also (Fig. 3). This bony invasion of the fracture lines may be attributed to the very high osteogenic potentialities of fibrous repair tissue, especially when formed in such sites as these. In the presence of such active bone formation it is difficult to conceive that union of the root fragments could ever take place except by bony ankylosis, but even this is to some extent precluded in the present case by the extreme mobility of the coronal root fragments.

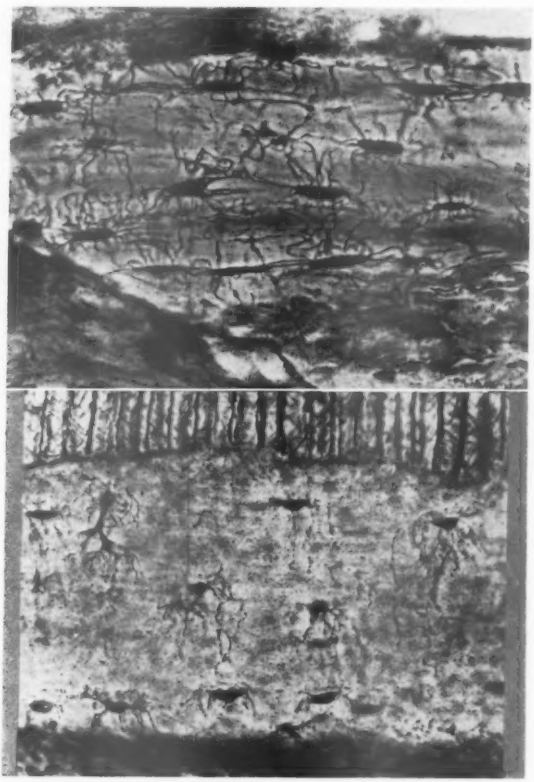
Mechanical injury to the tooth, whether or not it is of sufficient magnitude to cause actual fracture of the roots, usually produces a characteristic change in the cellular constituents of the pulp. This change has been referred to as a "fibrosis" of the pulp, and the replacement of the pulps of the present teeth by cells resembling connective tissue cells or fibroblasts would appear to correspond with this description. In spite of the change in the cellular constituents of the pulp, however, both teeth reacted to thermal stimulation, but this cannot be considered unusual as it has been estimated that a positive reaction to vitality tests is restored in over 70 per cent of fractured or injured teeth.[2, 3] The apical nerve fibers in the two teeth under examination certainly appear to be intact, but the state of the finer fibers, particularly in the coronal portions of these teeth, was not ascertained. It is not known whether this restoration to function represents a survival of part of the pulpal nerve supply or its regeneration following injury.

Like other highly specialized tissues, the dental pulp is particularly susceptible to injury and, except under certain well-defined conditions, does not regenerate. It can, therefore, be readily appreciated that there has been an abrupt cessation of tubular dentine formation as a result of injury to the tooth, but the cellular calcified collagenous tissue which is subsequently formed by the injured pulp demands further explanation. Previously this tissue has been described under a variety of names, such as secondary dentine, cellular dentine, osteodentine, osteocementum, secondary cementum, or calcific repair tissue, and the cellular calcified layer which has formed in the pulp chambers of

the present teeth appears to correspond closely with these descriptions. A closer examination of this tissue, however, reveals some of the essential features which serve to distinguish it from bone and cementum with which it has so frequently been confused.

One of the more obvious differences is that this tissue lacks any definite incremental pattern of growth comparable to that of bone, cementum, or normal tubular dentine. This may be attributed to the loss of odontoblasts, for secondary tubular dentine, when formed in response to a pulpal injury, always possesses an incremental growth pattern similar to that seen in primary dentine.

A.



B.

Fig. 9.—*A*, Typical osteocytes in a newly formed fragment of lamellar bone in the pulp of the left maxillary incisor. (Picrothionine stain. Magnification, ×475; reduced ¼.)
B, Apposition of cellular cementum to a previously resorbed area of root dentine. Note the appearance of the cementocytes. (Picrothionine stain. Magnification, ×475; reduced ⅛.)

Second, the cell inclusions in this tissue differ markedly from those of bone or cementum. They are fewer in number and less evenly distributed throughout the matrix, and they show a greater variation in size and shape

(Fig. 5, *B*) than is ever observed in the adult osteocyte (Fig. 9, *A*) or cementocyte (Fig. 9, *B*). Such variation has resulted, no doubt, from the inclusion of two or more cells within the same lacuna, as was observed in the hematoxylin-and-eosin-stained sections of the present teeth. The processes of the cell inclusions, in contrast to those of the osteocytes or cementocytes, are coarser and more irregular and often terminate blindly in the surrounding matrix. It has been assumed from observations in this study and in a previous study[4] that some of the cell inclusions, especially those close to the original primary dentine, are actually odontoblasts that have become trapped in the developing matrix. This assumption was based on the fact that some of the cell inclusions have definite tubular connections with the free ends of the adjacent dentine tubules (Fig. 5, *B*), and it would appear unlikely that any cell other than an odontoblast could maintain protoplasmic connections with existing dentine tubules. It is thought, therefore, that the cells of the pulp responsible for production of dentine matrix are less easily damaged by the effects of injury than the specialized odontoblasts, so that matrix formation is continued in spite of the gross upset of the odontoblast layer. Some of the damaged odontoblasts, together with other cells of the pulp, are therefore trapped haphazardly in the growing matrix; this accounts for the very uneven distribution of the cell inclusions in this tissue.

A final point in relation to the cellular calcified layer—a point that was observed in both of the present teeth—is its tendency to return to a modified form of tubular dentine. Formation of tubular dentine necessitates the presence of odontoblasts, so it is assumed that the new odontoblasts must have differentiated from the mesenchyme cells of the damaged pulp. This return to tubular dentine formation is more frequently seen in young teeth,[4, 21] and the fact that it usually commences in the apical portion of the root canals may be associated with the more rapid re-establishment of the blood supply to this part of the tooth following injury.

From the foregoing observations, it would appear that the cellular calcified tissue formed by the pulp in response to injury, although it resembles bone or cementum, is completely lacking in the organization of cellular elements which characterizes these tissues. On the other hand, the nature of the cell inclusions of this calcified tissue and its tendency to return to a modified form of tubular dentine serve to identify it more closely with dentine and therefore warrant its recognition as "cellular dentine."

SUMMARY

Two fractured central incisors removed six months after injury, together with their supporting alveolar bone, were examined microscopically. The reparative processes in relation to the fractures are described, and the nature of the cellular calcified tissue formed by the injured pulp is discussed. The closer identification of this tissue with dentine would appear to warrant its recognition as "cellular dentine" and serve to distinguish it from bone and cementum with which it is frequently confused.

The author wishes to thank the Surgical Department of the Royal Dental Hospital of London for the clinical material, the Radiology and Photographic Departments for the reproduction of the radiographs, and Mrs. C. Beck and Mr. J. Mercer for the preparation of the histologic material.

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Section by the

ARMED FORCES AND PUBLIC HEALTH SERVICE

STUDIES OF THE ORAL CIRCULATION

I. Preliminary Report on External Carotid Arteriography

Philip Person, D.D.S., Ph.D., Jerome Dubowy, M.D., and Seymour Zeff, D.M.D., Brooklyn, N. Y.

INTRODUCTION

MANY problems in oral pathology would be better understood if we possessed a greater knowledge of the physiology and pathology of the oral circulation. Until now, most studies of the oral circulation have been made with various injection techniques for filling the circulatory system of autopsy material. It is difficult, and very often impossible, to gain from such studies any real understanding of the dynamics of circulatory phenomena in the living organism. The Special Dental Research Laboratory of the Veterans Administration now has underway a program of studies designed to develop methods for the study and evaluation of the oral circulation in the *living* organism. This article summarizes some beginning experiences with the method of external carotid arteriography.

ARTERIOGRAPHY

In 1927, Egas Moniz,[1] the Portuguese neurosurgeon, discovered that the vascular system of a living animal could be visualized by means of x-rays if a solution of a radiopaque substance was injected into the blood stream of the animal. He also showed that filling and emptying of vessels and passage of the opaque materials through the vascular system could be followed by means of serial roentgenograms. Moniz thereby created present-day arteriography and angiography, techniques which have assumed major roles in the study of the circulation in almost all parts of the body.

Clinical arteriography and angiography are never performed unless a grave problem exists with definite indications for their use, for serious complications may arise in a small percentage of cases.[2] Nonetheless, these techniques are

From the Special Dental Research Laboratory, Radiology Service, and Dental Service of the Veterans Administration Hospital, Brooklyn, New York.

often indispensable and are used regularly in evaluating neurological, neurosurgical, cardiovascular, peripheral vascular, head and neck, and other problems. To our knowledge, there are no reports in the dental literature of arteriographic or angiographic studies of the oral circulation. This is not surprising since, in view of possible complications, the dentist rarely has felt compelled to request such procedures, although there are problems in oral medicine which would profit greatly from exploration by these methods. We have also been unable to find reports of arteriographic or angiographic studies of the oral circulation of animals.

This article describes efforts undertaken to determine the potential usefulness of arteriography in studies of the oral circulation. We were particularly interested in knowing whether the relatively narrow caliber of many oral vessels and the denseness of adjacent and overlying bone would decrease contrast to the extent that visualization of blood vessels on roentgenograms would be significantly impaired. Our results have indicated a definite usefulness of arteriography for clinical and experimental studies of the oral circulation. The present preliminary report has been prepared for the purpose of bringing this to the attention of our colleagues, especially in the field of hospital dentistry.

METHODS AND RESULTS

1. *Clinical Arteriograms.*—For studies of the human oral circulation, carotid arteriograms in our hospital files were reviewed. Most had been made for neurological or neurosurgical diagnostic purposes. In many instances, only the internal carotid circulation is of interest to the neurologist or neurosurgeon, so that the external carotid artery and its branches are bypassed. In other instances injections may be made into the common carotid or external carotid arteries. Those films in which the external carotid artery and its branches were visualized were selected for further study. Every hospital has such films, which represent a rich source of material for study by the dentist.

A typical arteriogram, in which the branches of both external and internal carotid arteries are visualized, is shown in Fig. 1. A diagrammatic tracing of the arteriogram is shown in Fig. 2. Beginning with the lowermost portion of the external carotid artery and proceeding cephalad, the following major branches were easily distinguished on the original film: the superior thyroid, lingual, external maxillary, occipital, internal maxillary, anterior deep temporal, and infraorbital arteries; also, the sublingual, submental, inferior and superior labial, angular, inferior alveolar, posterior superior alveolar, and infraorbital arteries. In addition, vessels of lesser caliber, not easily identified, are visible throughout. In Figs. 3 and 4 another arteriogram and an accompanying diagrammatic tracing are shown.

It must be stressed that positive identification of vessels may be hampered because of an inability to visualize structures in three dimensions. Thus, when superficial and deeper vessels course in the same area, confusion may exist concerning their identity. The identification of some of the smaller arteries in

Figs. 2 and 4 is therefore open to question. Also, we have not attempted to trace all the distal branches of the external carotid artery.

From the preceding, it is evident that many components of the arterial supply to the oral cavity may be visualized in the living human being by means of arteriographic techniques. When it is remembered that the films thus far studied have been made for nondental purposes and without specific interest in the oral circulation, it would appear that the method holds great promise. The use of stereoscopic roentgenograms, for which equipment now exists, would assist materially in identification of smaller arteries.

Fig. 1.

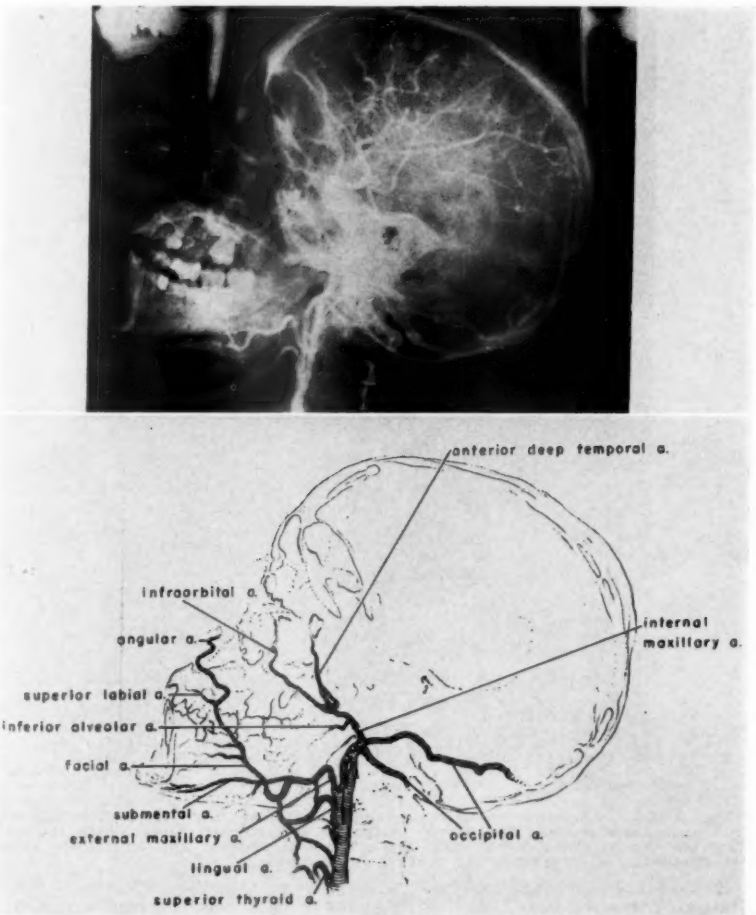


Fig. 2.

Figs. 1 and 2.—Arteriogram made by injection of the common carotid artery and showing both external and internal carotid circulations. In the diagrammatic tracing (Fig. 2), only branches of the external carotid are shown.

2. *Animal Arteriograms.*—In animal experiments, each dog was anesthetized with Nembutal and the common carotid artery was exposed. The animal was then positioned and preliminary roentgenograms were made to ensure proper

Fig. 3.

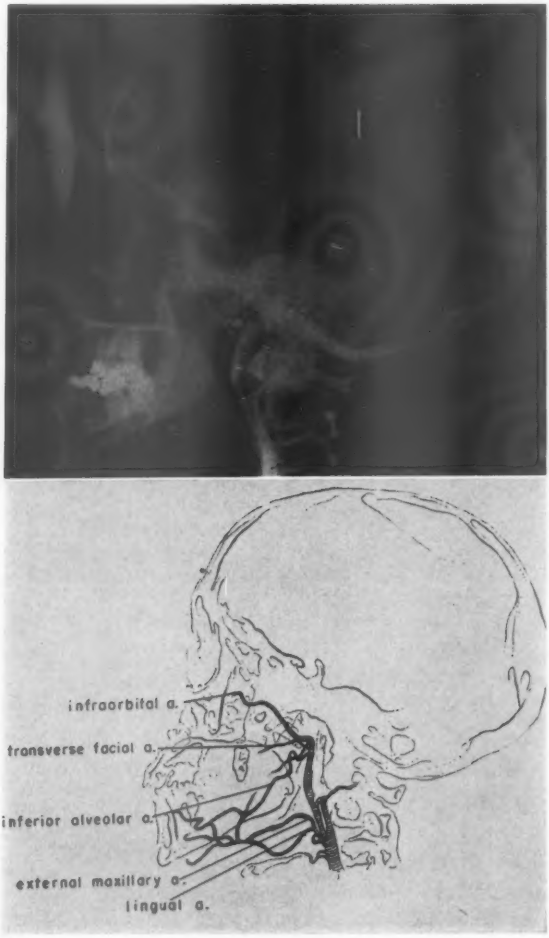


Fig. 4.

Figs. 3 and 4.—Arteriogram and diagrammatic tracing made from it. Only external carotid branches are shown. The uneven outline of the external carotid above the levels of origin of the lingual and external maxillary arteries as seen in Fig. 3, is often associated with atheromatous or arteriosclerotic changes in a vessel.

angulation, exposure time, etc. A 22-gauge needle was inserted cephalad into the common carotid artery and fixed in position by means of ligatures and clamps. Care was taken not to obstruct the flow in either direction. At a given

signal, serial roentgenographic exposures were begun with a Fairchild camera at one-half-second intervals. One second following this signal, rapid injection of 20 ml. of the contrast medium (50 per cent solution of Hypaque*) was made. Exposures were continued during and following the injection. A typical experiment is summarized in Figs. 5 to 11.

Fig. 5.

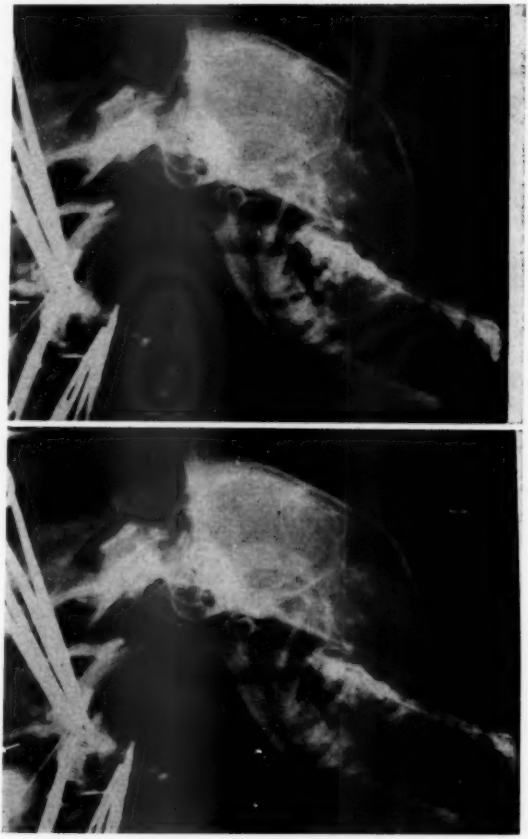


Fig. 6.

Figs. 5 through 11.—Serial roentgenograms taken at one-half-second intervals depicting the passage of injected contrast medium through the carotid arteries of the dog. Fig. 8 shows a tracing of the arteriogram shown in Fig. 7. Note the spasm of the internal carotid and the artery adjacent to it. This is thought to be a reflex response, defensive in nature, to the injection. For descriptions of the individual figures, see text.

(Figs. 7 to 11 on following pages.)

The sequence of events in passage of the contrast medium through the carotid circulation was as follows: As the injection of contrast medium was made, although the needle pointed cephalad, flow of the solution was toward the

*Hypaque is a product of Winthrop Laboratories, New York, N. Y.

heart and away from the head, *against* what is presumably the customary direction of blood flow in the artery (Fig. 5). One-half second later, as shown in Figs. 5 and 6, this flow away from the head was still maintained and the artery still had not filled in its customary direction of flow. The superimposed white arrows in Fig. 6 indicate the direction of flow. One-half second later (Fig. 7) *cephalad* flow had begun, and now the external carotid and internal carotid ar-

Fig. 7.

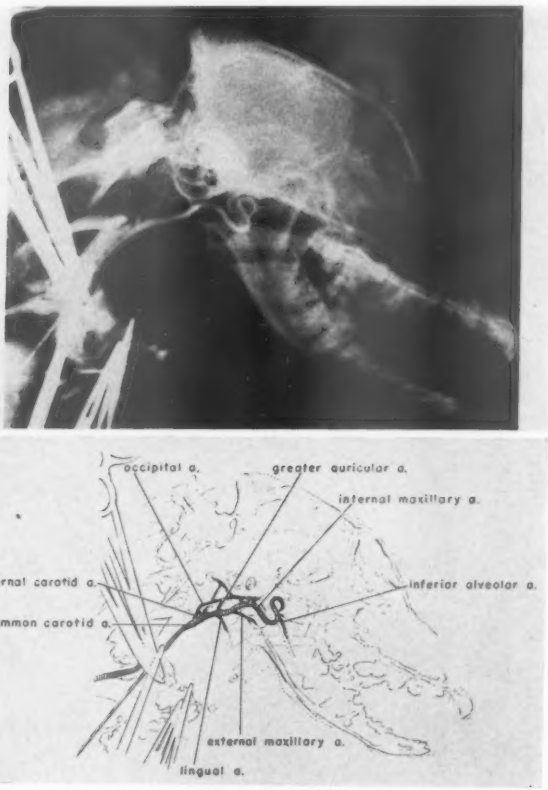
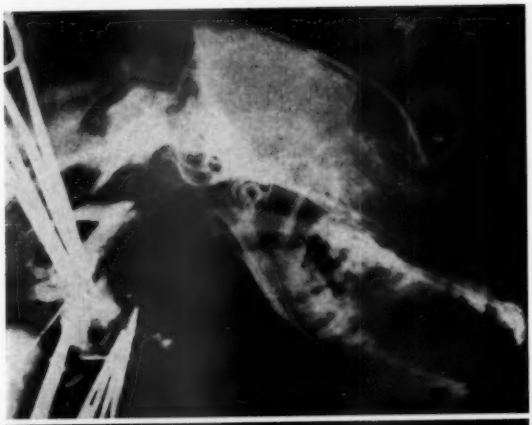


Fig. 8.

(For legend, see page 375.)

teries were visualized. Also defined were the following branches of the external carotid: the greater auricular artery, the lingual and external maxillary arteries, and the internal maxillary artery and its branch, the inferior alveolar artery. Fig. 8 shows a diagrammatic tracing of the photograph shown in Fig. 7. The internal carotid artery, as seen in Fig. 7, presents a most interesting outline. As it leaves the common carotid artery, it is of large caliber, but it soons narrows precipitously to an extremely fine caliber. This is most likely a result of spasm, or it may reflect the existing morphology. Were this a result of arterial spasm,

Fig. 9.



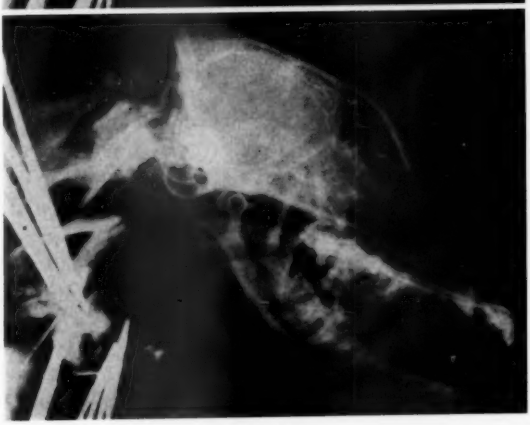


Fig. 10.

Fig. 11.

(For legend, see page 375.)

and had the external carotid artery also been in spasm, the resulting resistance to forward flow might explain the backward flow of contrast medium noted in Figs. 1 and 3. Reflex spasm of the internal carotid artery in response to injections is a well-known phenomenon.[3, 4] We have learned from neurosurgeons that a similar backflow of contrast medium is seen at times in human beings. In Fig. 9 the large caliber vessels have emptied, and only the lingual, external maxillary, and mandibular arteries are seen. One-half second later only a portion of the lingual artery is still visible, as seen in Fig. 10. In Fig. 11 all vessels have emptied.

DISCUSSION

It is evident from the studies presented here, that carotid arteriography and, hence, angiography are capable of yielding valuable information concerning the physiology of the oral circulation in living human beings and in experimental animals. In addition, data bearing on pathologic physiology and anatomic pathology may be gained from such studies. For example, in Fig. 3 the filling of the external carotid artery is uneven at and above the level at which the lingual and external maxillary arteries take origin. Such unevenness in outline of a vessel is often associated with atheromatous or arteriosclerotic plaques and obstructions in the walls of the vessel. The detection and location of such lesions elsewhere in the body has often been accomplished by means of arteriographic studies. The possibility of their detection in an area to which, for many reasons it is difficult to gain access in autopsy material offers exciting possibilities for gaining a deeper understanding of many problems in oral pathology.

SUMMARY

Studies of the physiology of the oral circulation, using the method of arteriography, have been made. Their purpose was to learn whether or not the technique could be applied successfully in areas where dense bone shadows are superimposed on small-caliber vessels. It was demonstrated that external carotid arteriography in human beings and in experimental animals holds much promise for obtaining data dealing with the physiology and pathology of the oral circulation.

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CENTRAL NEUROFIBROMA

Report of a Case

Harvey S. Johnson, Captain (DC) USN, Gordon T. Wannamaker, Lieutenant Colonel, MC, USA, James J. Humes, Lieutenant Commander (MC) USN, and Calvin W. Thompson, Captain, DC, USA*

TUMORS of the mandibular branch of the trigeminal nerve within the substance of the mandible are rare. Cornell and Vargas[1] reported an intraosseous neurofibroma, and Goldman[2] reported a neurinoma of the mandible.

The complexity of neurogenic tumors of the fibrous type promotes a somewhat confused nomenclature due to the intimate relationship of the connective nerve sheaths. Precise identification is particularly difficult when it is realized that a tumor could not arise from one of these intimately associated layers without involving the others. These tumors, being true neoplasms, have a tendency to recur following attempted removal.

Neurofibroma, neurogenic fibroma, neurogenic fibroblastoma, or perineural fibroblastoma, being names for the same entity, are in themselves not considered rare. In the oral cavity they generally arise from the trigeminal nerve branches in the tongue and palate, being less superficial than simple fibroma.

The etiology of neurofibroma is unknown, but trauma, endocrine dysfunction and/or a disturbed embryogenesis are implicated. Von Recklinghausen's disease, being a form of peripheral neurofibromatosis, also is considered as familial and inheritable.

The following case of a central neurofibroma of the inferior alveolar nerve is presented because of its rarity in location and recurrence.

CASE REPORT

A 34-year-old healthy-appearing Caucasian woman was first seen by us on Sept. 15, 1955. The chief complaint was paresthesia of the left side of the lower lip and tenderness of the dentition of the left mandible during mastication.

Hospital records revealed that the patient was referred to this hospital on March 4, 1955, for evaluation and treatment of a 2.5 by 1.5 cm. roentgenographic radiolucency posterior to the left second molar (Fig. 1), which was first observed during a routine dental roentgenographic examination. On March 5, 1955, the area of concern was surgically explored. A solid tumor mass which encompassed the neurovascular bundle of the inferior alveolar nerve was discovered. The tumor was enucleated without difficulty, leaving the

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nerve bundle intact. The contiguous bone was granular in appearance. The enucleated tissue was of a hard, nodular consistency. The histopathologic diagnosis was neurofibroma (Fig. 2). A careful and detailed family history failed to reveal any significant familial or congenital background of bone or cartilage disease.

Fig. 1.—Osseous lesion of left mandible as originally observed in March, 1955. Note the course of the mandibular canal. The left mandibular third molar is congenitally absent.

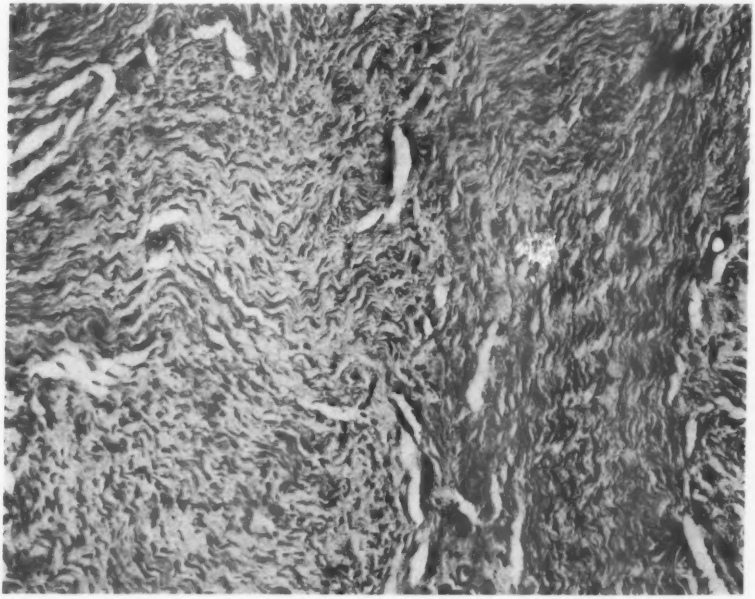

Fig. 2.—Photomicrograph of original excision specimen (March, 1955). The interlacing bundles of elongated spindle cells with wavy nuclei are clearly seen.

On Sept. 15, 1955, the patient was observed for a routine follow-up evaluation. Oral examination revealed a normal complement of teeth in a state of excellent repair and hygiene. The four third molars were missing. Periapical dental and extraoral roentgenograms revealed the radiolucent void of previous surgery. The architecture of the bone inferior to the first and second molars showed slight radiolucency.

Physical examination revealed, in addition, two brownish pigmented areas on the skin, one over the left buttock and one over the right scalp. Neither was associated with underlying collagenous or neurofibromatous changes. Chest roentgenograms were normal, and routine laboratory studies were within normal limits.

Fig. 3.

Fig. 4.

Fig. 3.—Roentgenographic presentation of osseous lesion of left mandible in April, 1957.
Fig. 4.—Postoperative roentgenogram of left mandible.

From September, 1955, to April, 1957, the patient was observed at approximately three-month intervals. The original symptoms of numbness of the left lower lip and tenderness of the dentition of the left mandible, particularly during the act of mastication, persisted. Repeated roentgenographic studies were inconclusive. Vitality tests of the mandibular teeth proved to be positive on repeated occasions. In January, 1956, a biopsy of the radiolucent space of previous surgery was performed. Histologic examination revealed only scar tissue. The intramuscular administration of 1 mg. of vitamin B_{12} daily for twelve days gave minimal relief of symptoms. Consultation and evaluation by the

neurological, neurosurgery, pathologic, radiologic, and medical services were invited. A thorough work-up by the endocrine clinic, including the laboratory studies of serum calcium, serum inorganic phosphorus, serum potassium, serum sodium, sedimentation rate, C-reactive protein, and a tracer dose of I^{131} were all reported within normal limits.

During a routine follow-up examination in April, 1957, a slight swelling was noted over the left mandible. At this time, roentgenographic studies revealed the formation of a definite radiolucent area of the left mandible involving the apices of the second molar roots and a perforation of the buccal plate just posterior to the second molar (Fig. 3). Recently the original symptoms had increased to such an extent that the application of lipstick caused pain over the left side of the lower lip. The patient was admitted to the hospital and on May 8, 1957, after induction of nasoendotracheal anesthesia, a surgical exploration of the left mandible was accomplished.

Fig. 5.—Photomicrograph of recurrent neurofibroma of left mandible. Note the similarity to the original tumor. (Magnification, ×100.)

As the bucco-alveolar periosteum was being elevated, a fibrous band of tissue was noted emerging from a smooth-margined perforation in the buccal cortical plate of the left mandible and passing into the soft tissue. This band and a portion of the buccal tissue were excised. The two molars and the buccal cortical plate from the first molar area posteriorly to the retromolar area and extending inferiorly to the mandibular canal were removed. The field of concern was adequately visualized and a hard nodular tumor involving the inferior alveolar nerve was easily enucleated (Fig. 4). The soft tissues on the medial aspect of the ascending ramus were reflected to expose the lingula. The inferior alveolar nerve was identified, clamped, and cut. The nerve proximal to the tumor was extirpated. A similar procedure was accomplished with the distal portion of the nerve at the mental foramen.

The pathology report revealed "central neurofibroma" (Fig. 5).

COMMENT

The first problem in this case, on evaluation in September, 1955, was diagnosis. It is accepted that a neurofibroma can and does recur. The principle of biopsy when the diagnosis of an existent lesion is in doubt was followed in January, 1956. In consideration of tooth vitality, a lack of abnormal laboratory studies, inconclusive roentgen findings, and a negative biopsy, the symptomatology was diagnosed as nerve-tissue trauma, due to initial surgery in March, 1955. However, recurrent tumor could not be discounted.

When definite roentgen evidence of tumor was observed together with the clinical feature of a swelling of the buccal tissue and the extended period of symptomatic history, surgery was indicated for removal of the neurogenic tumor and that segment of the mandibular nerve confined within the mandible.

The abnormal course of the mandibular canal, passing superiorly to the area of the congenitally missing third mandibular molar at which point the original neurofibroma was removed, is worthy of note. It may appear that a disturbed embryogenesis was the etiological factor of the first neurogenous tumor, as trauma had been denied and endocrine dysfunction had been disproved. One wonders whether the second or recurrent tumor was a result of the original faulty embryogenesis or whether it was caused by the trauma of the initial surgical procedure.

CONCLUSION

1. A case of a central neurofibroma within the left mandible is reported.

2. Etiology of the tumor in this case appears to be one of disturbed embryogenesis.

3. The neurofibroma may recur after enucleation.

4. Once a neurogenous tumor recurs, the removal of the parent tissue is justified to prevent a possible recurrence which may manifest malignant changes.

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ANNOUNCEMENTS

American Academy of Dental Medicine

The Academy will hold its thirteenth annual meeting at Hotel Claridge in Atlantic City, New Jersey, May 14 to 17, 1959. The meeting will feature discussions of the role of oral medicine in the practice of dentistry.

Among those scheduled to lead the discussions are the following: Emanuel Cheraskin, Professor and Chairman, Section on Oral Medicine, University of Alabama School of Dentistry; Lester R. Cahn, Associate Professor, Chairman of the Division of Oral Pathology, Columbia University School of Dental and Oral Surgery; S. Sigmund Stahl, Assistant Professor, Department of Periodontia and Oral Medicine, New York University College of Dentistry; Harry Blechman, Associate Professor and Chairman, Department of Microbiology, New York University College of Dentistry; Austin H. Kutscher, Research Associate and Lecturer in Clinical Pharmacotherapeutics, Division of Stomatology, School of Dental and Oral Surgery, Faculty of Medicine, Columbia University; Harold J. Jeghers, Professor of Medicine, Seton Hall College of Medicine and Dentistry; Bernard W. Greenberg, Assistant Professor and Chairman of the Department of Endodontics, Fairleigh Dickinson University School of Dentistry; Morris B. Auerbach, Associate Professor and Chairman, Department of Endodontics, New York University College of Dentistry; and George G. Stewart, Assistant Professor of Endodontics, Temple University School of Dentistry.

A special program of social events and sight-seeing has been arranged for registrants and their families.

All Academy members, as well as other interested dentists and physicians, are cordially invited to attend. For programs and reservations, address Dr. Harold A. Lentz, 619 Main Ave., Passaic, New Jersey.

Tenth Annual Berkshire Conference in Oral Pathology and Periodontology

Tufts University School of Dental Medicine will conduct the tenth annual Berkshire Conference in Oral Pathology and Periodontology at Eastover in Lenox, Massachusetts, June 14 to 18, 1959. The Conference will consist of four days of lectures, seminars, and panel discussions by outstanding teachers and clinicians in dentistry and medicine and their related fields.

The central theme of the Conference will be "Trauma and Oral Disease." There will be a closed-circuit television session devoted to a step-by-step presentation of the technique of occlusal equilibration.

The program will include the following:

Col. Joseph L. Bernier, Armed Forces Institute of Pathology, Washington, D. C.
A Clinico-Pathological Seminar Devoted to "Trauma in the Causation of Oral Mucous Membrane Disease."

Dr. Basil G. Bibby, Eastman Dental Dispensary, Rochester, New York. THE ORAL BACTERIAL FLORA, ITS SIGNIFICANCE IN THE MANAGEMENT OF PERIODONTAL DISEASE; CALCULUS FORMATION.

Dr. Milton B. Engel, College of Dentistry, University of Illinois, Chicago, Illinois. THE CONNECTIVE TISSUE GROUND SUBSTANCE, ITS SIGNIFICANCE IN DISEASE OF THE GINGIVA AND UNDERLYING TISSUE.

Dr. Irving Glickman, Tufts University School of Dental Medicine, Boston, Massachusetts. TRENDS IN PERIODONTOLOGY DURING THE LAST DECADE. PATHWAYS FOR FUTURE DEVELOPMENT.

Dr. Balint J. Orban, Loyola University School of Dentistry, Chicago, Illinois. TRAUMA AND THE PERIODONTIUM.

Dr. Sigurd P. Ramfjord, School of Dentistry, University of Michigan, Ann Arbor, Michigan. A PRACTICAL CLINICAL APPROACH TO THE ELIMINATION OF OCCLUSAL TRAUMA BY OCCLUSAL EQUILIBRATION.

Dr. Louis Weinstein, Tufts University School of Medicine, Boston, Massachusetts. THE ACTION OF ANTIBIOTICS UPON MICROORGANISMS, TISSUES AND THE GENERAL WELFARE OF THE PATIENT.

For further information and application, write to Tenth Annual Berkshire Conference, Tufts University School of Dental Medicine, 136 Harrison Ave., Boston 11, Massachusetts.

First Annual Pacific Coast Oral Pathology Workshop

In the past several years oral pathologists and others interested in this field on the Pacific Coast have been meeting informally to discuss the possibility of an annual program devoted to oral pathology and the establishment of study groups in the larger cities. The support and encouragement of the American Academy of Oral Pathology to this effort have been given without qualification.

This workshop will be held just prior to a state meeting on May 3, 1959. The workshop will take up the subject of "Periapical Lesions" will consist of a day devoted to the presentation of short, pertinent papers as a background for presentation of prepared cases by a panel of oral pathologists and clinicians. The complete program will be made into a syllabus for the use of all participants. A nominal registration fee of approximately $25.00 to defray the cost of the workshop will be made.

The meeting place will be either the University of Southern California Dental School or the College of Medical Evangelists Dental School in Loma Linda. The final program and its participants will soon be announced.

If you have any suggestions or any questions concerning the workshop, please communicate as soon as possible with Dr. Weldon Bullock or Francis V. Howell, Department of Pathology, University of Southern California School of Medicine, 1200 North State St., Los Angeles, Calif.

Austrian Dental Congress

The annual Austrian Dental Congress will be held May 20 to 23, 1959, in Graz, Austria.

The main subject will be "Esthetic Problems in Dentistry and in Maxillofacial Surgery." A secondary subject, "The Preservation and Amputation of the Pulp," will also be taken up.

Further information may be obtained from the president of the Congress, Prof. Dr. Richard Trauner, Zahnklinik und Kieferstation, Auenbruggerplatz 12 (Landeskrankenhaus), Graz, Austria.

Southwest Society of Periodontists

The fourth annual meeting of the Southwest Society of Periodontists will be held on May 1 and 2, 1959, at the Gunter Hotel in San Antonio, Texas. The clinicians will be Drs. E. Cheraskin and R. Gottsegen.

For further information, please contact Dr. Claude Nabers, 1014 South Texas Bldg., San Antonio, Texas.

Boston University and Massachusetts Memorial Hospitals

The Department of Stomatology, Division of Graduate Studies, School of Medicine, Boston University, and the Massachusetts Memorial Hospitals announces the following postgraduate courses:

Advanced Course in Periodontal Therapy. Drs. Henry M. Goldman, Bernard S. Chaikin, and Staff. March 2-7, 1959. $250.00.

Technics in the General Practice of Dentistry. Staff. March 9-14, 1959. $175.00.

Periodontal Therapy. Drs. Henry M. Goldman, Bernard S. Chaikin, and Staff. March 16-27, 1959. $350.00.

Tooth Preparation and Elastic Impression Material Technics in Crown and Bridge. Dr. Arthur Kahn. March 30—April 1, 1959. $100.00.

Full Denture Prosthesis. Dr. Chester Landy. April 6-10, 1959. $150.00.

Occlusal Adjustment. Drs. Bernard S. Chaikin, Henry M. Goldman, and staff. April 27—May 1, 1959. $200.00.

Endodontics. Dr. Herbert Schilder. May 11-15, 1959. $175.00.

Periodontal Prosthesis. Drs. Leo Talkov and David J. Baraban. May 18-22, 1959. $200.00.

Surgical Technics Employed in Periodontal Therapy. Dr. Lewis Fox. May 25-29, 1959. $200.00.

Pulp Protection in Operative Dentistry. Dr. Harold Berk. June 4-6, 1959. $100.00.

Periodontal Pathology. Drs. Henry M. Goldman, Jack Bloom, and Staff. June 8-12, 1959. $250.00.

Detailed hour-by-hour rosters for the courses are available upon request.

For further information or for application blanks, apply to Director, Postgraduate Courses, Department of Stomatology, Massachusetts Memorial Hospitals, 750 Harrison Ave., Boston 18, Massachusetts.

University of Illinois

The College of Dentistry is accepting applications for two- and three-year full-time courses in oral surgery, beginning in September of 1959. Both programs carry credit toward a master of science degree.

Further information may be obtained from Dr. Daniel M. Laskin, Department of Oral and Maxillofacial Surgery, University of Illinois, 808 S. Wood St., Chicago 12, Illinois.

New York University

A course in "Development of Effective Practice" will be given June 22 to 26, 1959, at the College of Dentistry. The course consists of lectures and participation sessions to

teach the dentist and his staff how to motivate patients to accept more effective dental service. Training the secretary and other components of management are included. Instructors will be Dr. J. Lewis Blass and his associates.

Further information may be obtained by writing Postgraduate Secretary, New York University College of Dentistry, 421 First Ave., New York 10, New York.

University of Minnesota

The second annual course in advanced oral pathology will be given during the week of April 20 to 24, 1959. The course consists of both lectures and laboratory work. The subjects and faculty will be as follows:

Tumors of the Skin. Dr. Herbert Z. Lund.
Tumors of Soft Tissues. Dr. Raffaele Lattes.
Skin Dermatoses. Dr. Robert Goltz.
Non-neoplastic Diseases of the Jaws. Dr. William Shafer.
Tumors of Bone. Dr. David C. Dahlin.
Radiation Pathology. Dr. Donn Mosser.
Advanced Roentgenographic Diagnosis. Dr. Edward Stafne.
Salivary Gland Pathology. Dr. Anand Chaudhry.
Odontogenic Lesions. Dr. Robert Gorlin.

The tuition will be $65.00. The course is limited to fifteen persons. Housing for the five days will be approximately $15.00. Those interested should immediately contact Dr. Robert J. Gorlin, Chairman, Division of Oral Pathology, School of Dentistry, 242 Owre Hall, University of Minnesota, Minneapolis 14, Minnesota.

University of Oregon Dental School

A course in "Oral Pathology and Diagnosis in General Practice" will be given April 27 and 28, 1959, from 9 A.M. to 5 P.M. Instructors will be Robert J. Gorlin, Chairman, Division of Oral Pathology, University of Minnesota School of Dentistry, and Norman H. Rickles, Head, Department of Oral Pathology, University of Oregon Dental School. There will be a $40.00 fee for the course.

University of Pennsylvania

The following postgraduate courses are announced by the School of Dentistry:

EXTRAORAL TRACERS IN REHABILITATION OF FUNCTION. March 16 to 18, 1959. Dr. Ernest R. Granger.

PULP REACTIONS TO OPERATIVE PROCEDURES. April 2 and 3, 1959. Drs. Samuel Seltzer, I. B. Bender, and Louis I. Grossman.

MINOR TOOTH MOVEMENT AND TEMPORARY STABILIZATION. April 8 to 10, 1959. Dr. Leonard Hirschfeld.

CLINICAL OCCLUSION. April 20 to 24, 1959. Dr. Arne G. Lauritzen.

ENDODONTICS. April 13 to 24, 1959. Dr. Louis I. Grossman.

COMPLETE DENTURE PROSTHESIS. April 27 to May 1, 1959. Dr. M. M. DeVan.

NEWER CONCEPTS IN ORAL REHABILITATION. May 4 to 8, 1959. Dr. Charles B. Sceia.

For further information and application, write Dr. Lester W. Burket, 4001 Spruce Street, Philadelphia 4, Pennsylvania.

Temple University School of Dentistry

Applications are now being accepted for the position of Research Fellow and Clinical Instructor in the Department of Endodontia at the Temple University School of Dentistry in Philadelphia.

The position offers the applicant an opportunity to pursue research projects related to endodontics, while at the same time instructing students in clinical endodontic techniques. Remuneration is $4,200.00 a year. The applicant should be available Aug. 1, 1959.

Applicants should send information as to background, interest, and clinical experience to Dr. Leonard Parris, Temple University School of Dentistry, Broad Street above Allegheny Ave., Philadelphia 40, Pennsylvania.

University of Tennessee College of Dentistry

The following postgraduate courses are announced:

Orthodontics for General Practitioner. March 18 to 20, 1959.
Endodontia. March 23 to 25, 1959.
Oral Surgery. March 30 to April 1, 1959.
Full Denture Prosthesis. April 17 and 18, 1959.
Operative Dentistry. May 25 to 27, 1959.
Periodontology. June 10 to 12, 1959.

Guest speakers, selected for their acknowledged leadership in their fields and for their teaching ability, have been invited to participate in the courses.

Further information may be obtained from the Postgraduate Department, 62 South Dunlap, Memphis, Tennessee.

Michael Reese Hospital Announces Dental Internships

Michael Reese Hospital announces that it is now receiving applications for its twelve-month dental rotating internships, starting July 1, 1959.

Those interested should write to Dental Department, Michael Reese Hospital and Medical Center, 29th St. and Ellis Ave., Chicago 16, Illinois.

The Michael Reese Hospital Department of Dentistry is approved by the American Dental Association Council on Dental Education and Hospital Dental Service.

Histopathology Study Club

Jan. 1, 1959, marked the first year of sponsorship of the Histopathology Study Club by the Department of Dentistry of Michael Reese Hospital and Medical Center.

Dr. Balint Orban, histopathologist for the Study Club, reported a successful year of operation. Monthly case histories were sent to the 110 members, including dentists and dental schools from coast to coast. According to Dr. Orban, the future of the club appears extremely bright. For the next year some very unusual human autopsy material is being prepared.

At present a limited number of memberships are still available in the Study Club. Anyone interested in participating in this interesting, beneficial, and educational effort may write to the Department of Dentistry, Michael Reese Hospital and Medical Center, 29th St. and Ellis Ave., Chicago 16, Illinois.